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ELECTRO-COMA THERAPY OF PSYCHOSES *

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INTRODUCTION

THE therapy of psychoses has advanced rapidly during the past decade. Observation and analysis, hydrotherapy and occupational measures, and symptomatic medication all have their fields of usefulness. However, there have been introduced more specific therapies which depend upon direct chemical and electrical influence upon brain function.* In 1932 Sakel [1] developed the insulin shock treatment for schizophrenia, a method which opened a new era in the therapy of psychoses. Shortly thereafter, the use of the metrazol convulsion treatment was the product of the research of von Meduna.[2] Metrazol, at first recommended for schizophrenia, proved more effective in depressed states. The results of this method were definitely valuable, but the psychologic discomforts and the orthopedic complications were major obstacles in its use. Recently, as an outgrowth of electrical brain study and the accepted use of convulsive therapy, the electro-shock methods were begun in 1938 by Cerletti and Bini.[3] Finally, during the last few years, a surgical procedure, frontal lobotomy, was established by the work of Moniz and was introduced into our country by Dr. Walter Freeman.[4, 5]

It is the purpose of this paper to record the clinical results in a series of 70 patients treated by the electrical method. This report includes a tabulation of the therapeutic results in the various types of psychoses and a consideration of the side actions and complications. Also mention will be made of several parallel research studies which have been undertaken. Finally, some interpretation will be offered of the results obtained in the light of the modern concepts of psychoses.

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Method and Material

The technic employed is similar to that which has been outlined by previous writers (Kalinowsky,[6, 7] Gonda,[8] Impastato and Almansi[9]). The reader may refer to these pioneer papers for a historical review of the subject and for technical details of the apparatus.

The method of treatment is briefly as follows: Electrodes are placed on the forehead of the patient. The apparatus is set at the desired current values. This usually means a dose of from 400 to 600 milliamperes at a time interval of from 0.2 to 0.5 second. The voltage for such a treatment may range from 60 to 150 volts. As the current is applied, unconsciousness occurs instantaneously. The patient becomes temporarily rigid in extension or he may flex forward. If the dosage has been small, there will be a few muscular twitchings only. If the dosage has been adequate, the individual will shake violently in a hard convulsive reaction lasting from 30 to 60 seconds. Following the convulsion, there is a period of stupor lasting minutes, from which the patient emerges gradually. The return to complete consciousness takes place by stages over a period of from minutes to half an hour or an hour.

Each patient receives a course of such treatments, the number of reactions depending upon the individual case. As a rule, from 6 to 12 treatments are necessary for a successful result. The treatments are given on alternate days, or usually three a week.

The material includes a group of private patients who, for the most part, showed evidences of early stages of mental disease. These patients were in the psychotic group; most of them had some type of depression and the remainder schizophrenia. In considering the therapeutic results, an attempt was made to separate the patients into various diagnostic headings. The commonly accepted groups of hebephrenic schizophrenia, paranoid schizophrenia, schizodepressions, simple depressions, agitated depressions, and involutional melancholia were used.

It must be pointed out that it is more difficult to classify patients who are in the earlier stages of mental disease than those whose illnesses are far advanced. Furthermore, one must realize that classifications are arbitrary and patients are real, and that the modern psychiatrist, using new methods of therapy, cannot accept the former classifications as rigid and final. Indeed, the point of view of Singer[10] deserves emphasis. He comments upon the difficulty of separating the various diagnostic groups. "It is always a problem not of all or none, but of more or less." It is Dr. Singer's thesis that the major symptoms, or the fact of the illness, are produced by the nature and location of the structural damage, whereas the form is determined by the person who is ill. Quoting from Singer: "Thus, one and the same organic lesion may give rise to a manic-depressive form of psychosis in one person and to a schizophrenic or paranoid psychosis in another." The surprising

results which have been obtained with the electrical method of treatment corroborate the modern point of view of Singer as against the rigid classifications of Kraepelin.

Terminology

Before mentioning the results obtained, it may be appropriate to present the terms which I have found helpful in discussing this subject. The term "electro-coma therapy" is proposed in place of the phrase "electro-shock treatment." The concept, shock, connotes either a painful and disagreeable electrical sensation or the vasomotor collapse which is part of "surgical shock." In this treatment the patient does not experience any dreadful electrical sensations nor does he show the circulatory collapse which is part of "surgical shock." The essential feature of this method is the period of unconsciousness, although convulsions are also a major feature. The term "electro-coma therapy" is descriptive of the method and does not connote false or unfavorable pictures to the patient and his family.

For the reactions which patients show, the terms "petit" and "grand mal" are used in the current literature. These terms have been introduced because of a certain resemblance to the manifestations of epilepsy. It has been my custom to use the phrase "minor reaction" for a brief episode of unconsciousness without a major convulsion. For the complete reaction with unconsciousness and convulsive state, the term "major reaction" is used. Occasionally the individual will have unconsciousness with marked rigidity and some twitching and shaking movements. For this in-between type of response, the term "moderate reaction" is used. The terms "petit" and "grand mal" have belonged to the concept of epilepsy for ages; likewise, the designation "petit mal" usually applies to a momentary state of hypoconsciousness and not to complete unconsciousness, which occurs with the electrical method. I have found the designations "minor," "moderate," and "major," as useful, accurate, and free of the epileptic association.

Clinical Appraisal of the Therapeutic Value

A detailed summary of the results obtained is included in the accompanying table. This shows the identification of the patient, the age, the duration of symptoms, the number of treatments, and the results obtained. One may summarize the details of the table with the brief statement that the changes resulting from the electro-coma treatment have been remarkable and the percentage of improvement high. Yet this improvement is somewhat selective, more prompt in certain types of patients than in others, and undoubtedly more permanent in certain patients. It may be of value for us to consider the therapeutic benefit from the standpoint of the group of patients treated.

A. The Combined Group of Involutional Melancholia and Agitated Depressions of Later Life. Within these categories are included those patients

whose ages range from 45 to 70 and whose major symptoms are those of depression. The composite picture of a patient in this group is a woman of about 60, previously cheerful, industrious, and capable, who has become sleepless, restless, troubled by visceral distress, and overwhelmed by a sense of catastrophe. Such an individual has been ill for many months, has lost considerable weight, has developed a sense of hopelessness, and has wished for death or has even made suicidal attempts.

The patients in this group respond remarkably well to electro-coma treatment. Eighteen patients belong to this group. All who were treated showed a favorable response. However, in three of the patients there are residual symptoms which indicate that the recovery has not been complete. Several of the patients were close to or above 70 years of age. They had been severely depressed for months. Two of the group had made suicidal attempts. They have shown a surprising recovery, with a return to well-being. Our results in this older age group corroborate the encouraging reports of Robinson,[11] who has shown the comparative safety of this method and the good therapeutic response of such patients to the electro-coma treatment:

T. V., a woman of 51, was sick for several months in the spring of 1941. She had been a cheerful and capable individual, happy in her home, sociable with friends, and interested in many activities. Gradually she lost the desire for company, could not eat, and became sleepless. Week after week her condition grew worse; she lost a great deal of weight, and was unable to read, eat, or sleep. She was overwhelmed by a sense of hopelessness. After two months of such distress, she attempted suicide by cutting her throat. First aid was given in an emergency hospital and she was then taken to the Post-Shaker Sanitarium. En route to the sanitarium she made another attempt at suicide by jumping out of the car.

She was observed in the hospital for several weeks. She refused all food saying: "It doesn't pay to eat, I want only to die. I can't eat; I don't have any bowel movements. My arms and hands are changed; my nerves are twisted in my belly and it seems as though they are moving around like worms. My brain doesn't function any more. There is no use, I am going crazy and no one can help me." These somatic delusions were intense and the patient was agitated with obvious suffering.

The electro-coma treatment was begun early in July. A series of 10 treatments was administered. During the course of the treatment, she became conversational, took an interest in her fellow patients, and remarked: "I am anxious to get my nerves collected and to get back to work. I know I am going to be happy." August 1 she stated: "These treatments have helped me a good deal. I know I am going to be well." During the early part of August she was somewhat elated and this cheerful mood continued for a while. The patient was discharged in September and resumed her position in her family. She has been seen several times during the fall of 1941 and has remained symptom free. Indeed, she has gained weight, renewed her usual acquaintances and activities, and is apparently well.

B. Depressions. It is certainly no easy matter to distinguish between endogenous depression and involutional melancholia. We have used the term, endogenous depression, for the cases of younger individuals whose family histories show a strong constitutional tendency to this illness. In several instances these patients have had previous attacks. In the group of

endogenous depressions there were 17 patients. The results in this group of patients have been excellent. Patients who had been ill for months or years responded favorably and quite promptly. As a rule, some six to eight treatments were sufficient. However, after this preliminary course, the patients were observed for a period of several weeks. If there was a tendency to relapse, additional treatments were given. In two instances, an additional course of six treatments was necessary.

A typical case history in this group is that of Mr. W. C.:

This patient was 40 years of age, a mechanic by occupation, unmarried, who had been ill for some eight months. A previous attack of depression occurred at the age of 20 and lasted a year and a half.

This individual had been a cheerful, self-confident, and capable person. Gradually he became morose, withdrew from his social contacts, and complained of weakness. The weakness was so profound that he could not work. Gradually the additional features of insomnia, self-accusation, and suicidal thoughts appeared. The patient believed that others were talking about him because of his sinfulness and that in his youth he had developed a habit which had brought about his ruin and from which he could never recover.

A course of six electro-coma treatments was given in a period of two weeks. Like a cloud moving away and revealing sunshine, so there was a remarkable change in this man's mood. The sad face and droopy posture gave way to an effervescent smile and erect, brisk walk. He looked and acted like a happy individual. Treatment was discontinued and he was allowed to go home on trial. Gradually he became sad, inactive, and once more obsessed by dark thoughts. The treatment was resumed and six more electro-coma reactions given. He was discharged in May of 1941 and has remained perfectly well during a period of six months.

C. Schizodepressions. There are many patients who show a definite combination of a markedly depressed mood with hallucinations and delusions, which are usually thought to be characteristic of schizophrenia. One who examines such a patient may be unable to determine whether the illness is a true schizophrenia or a depression. This doubt, when one faces such a patient, substantiates the statement of Dr. Singer which was quoted above. There are obviously individuals who cannot be classified as one or the other but who have an interweaving of mood change and schizophrenic features. The term, schizodepression, is used for such patients. Ten patients are included in this group. The typical patient in this group was a younger individual, often an intelligent and successful personality when well, and often without a family history of psychosis. These patients had active hallucinations and responded directly to them. There were voices which accused the individual of misdeeds or invited the person to some rendezvous. The accusations frequently revolved about the theme of sex. The members of this group did fairly well with electro-coma treatment. Seven have shown a favorable response whereas the improvement in the remaining three has been partial or temporary.

The following case history illustrates this type of patient:

Miss D. N. was a woman of 40, who had been an efficient secretary in a business concern for many years. Gradually the thought occurred to her that she was being

observed by her fellow employees and that one man in particular was sending messages to her. She believed that certain gestures represented messages of love. When she phoned him and he denied any attempts to arrange a rendezvous, she became incensed and then disheartened. She was quite bewildered by this apparent contradiction. Indeed, she not only interpreted the messages, but believed she could hear his voice talking to her. And yet he made a complete denial of any interest in her. She became depressed, could not sleep nor eat, and gave up her work. Saddened and bewildered, she felt there was no way out except death. She made two suicidal attempts. First she cut her wrists, but the bleeding was quickly stopped. Later she injected 360 units of insulin into herself. (She was accustomed to using the insulin syringe in the treatment of her diabetic mother.) After she had injected the insulin there was a change of heart and she immediately injected a concentrated sugar solution to counteract the effect of the insulin.

The patient was taken to the hospital. At first she was negativistic and inaccessible. Later she appeared to be hearing voices and she reported both optical and tactile hallucinations.

The electro-coma treatment was begun in June of 1941. After several treatments the patient became calm. After the ninth treatment, the hallucinations disappeared. The entire course consisted of 12 major reactions.

This patient has been followed (by correspondence) over a period of five months. She has made a splendid recovery, has resumed her place in the home, and is once more an efficient employee of her concern.

D. Manic Psychoses. Two patients, L. R. and L. J., with acute manic psychoses, were treated. Both showed a favorable reduction in the excitement as a result of the electrical treatment. However, in each instance the cessation of treatment was followed by a return of psychomotor restlessness and hypomanic behavior. A second short series of treatments was used, again with a calming influence. Both patients are now well.

E. Schizophrenia. The schizophrenic group undoubtedly encompasses a large assortment of patients. Any study of schizophrenia must differentiate between those individuals who have never achieved full and complete adjustment to life and those who had been successful but then developed serious evidence of illness. The prognosis in the former (dementia precox) is certainly far different from that of the latter.

Three patients with dementia precox were treated. Two of these have improved. However, the improvement is not complete and the prognosis remains unfavorable.

In the larger group of paranoidal schizophrenia the list includes 18 patients. Four of these patients have shown little or no improvement. Of these, three were considered advanced cases of schizophrenia. The remaining 14 have shown definite favorable changes as a result of electro-coma treatment. Indeed, practically every patient responded to electricity with an improved attitude and a cheerful mood response. The more serious chronic types of schizophrenia tend to relapse quickly. The others have continued to show progress. One of these patients was truly remarkable in that she had been sick for five years. This patient was treated in the spring of 1941, after several years' previous hospitalization and the failure of metrazol and

insulin shock therapy. Since her discharge from the hospital in April, 1941, this patient, F. R., has successfully maintained her place in society.

Treatment in the schizophrenic group was always begun with a definite attitude of skepticism. The malignant nature of schizophrenia is recognized. This malignant nature may account for the tendency to relapse after improvement by shock methods. The improvement which we have obtained with electro-coma treatment may, like that obtained with insulin, be but a pause in the course of the illness rather than a complete removal of the disease.

F. Miscellaneous Cases. In several instances, the electro-coma method was used for patients who did not belong to the above groups. For example, a patient with a severe compulsion neurosis was given a course of eight treatments. This was followed by a slight change in behavior, but there was a return of the symptoms when the treatment was stopped. A chronic alcoholic, who had periods of depression, was given several treatments. The mood change was corrected, but unfortunately the first stop this patient made when he left the hospital was his favorite bar.

The Psychologic Side Actions of Electro-Coma Therapy

Although the clinical results have been extremely satisfactory, yet certain side actions occur, usually temporary, which are quite disturbing. These side actions may be in the form of (1) marked amnesia, (2) periods of elation, and (3) episodes of confusion.

1. Amnesia. Following an electro-coma treatment, the return to consciousness is gradual, a step by step resumption of mental functions, and it may be a matter of minutes or several hours before the patient is fairly alert. Though awake, he is often bewildered, especially because he does not remember what had happened before, during, and immediately after the treatment. It is a common experience for a patient to have forgotten the fact that he has received a treatment. This amnesia is quite marked as regards the inability to recall time and place. The amnesia for events immediately preceding treatment is striking. As a rule, there is only a slight and transitory forgetfulness with the first few treatments. This amnesia is referred to as lacunar, i.e., a "spotty" memory disturbance. With each successive treatment the extent of the forgetfulness may become greater, as though each little spot of amnesia had been pooled into a large lake. Thus it is fairly common, particularly in older patients, for considerable blankness to develop after the sixth or eighth treatment. Such patients have forgotten how long they have been in the hospital, the names of the people with whom they are in contact, and many of the experiences of the past. It is remarkable that many of them have forgotten completely the dreadful, anxious, and suicidal thoughts they had before treatment.

There is a tendency toward a spontaneous recovery from such amnesia. When the patient is reminded of a certain event he can recall the situation

quite readily and he is apt to remember the episode from this point on. As a rule, patients recover from this memory disturbance in the matter of days or weeks. The longest instance of disturbed memory in this group of patients is that of Mrs. D. P., a woman of 62, who had received eight electro-coma treatments. She improved considerably and was, therefore, allowed to go home within several days following the last treatment. At home she could not remember many of the activities during the month or two before she entered the hospital. But her chief difficulty was that she had forgotten the cooking recipes with which she had been familiar. She was embarrassed when meeting old friends because she could not remember their names or certain social events that she had attended. Mrs. D. P. regained her memory, but it took a matter of some three months before she had fully recovered.

Our observations with regard to amnesia correspond with the favorable prognosis in memory disturbance reported by other workers (Ziskind,[12] Myerson [13]). For example, two of our patients, one a teacher and the other a research assistant, showed marked memory disturbance which lasted for several weeks. Both had made complete recoveries from serious mental illnesses, but were troubled by amnesia. In each case the return of memory was satisfactory. The research worker, whose job it was to translate foreign medical journals, stated that she had regained her normal speed and efficiency in this difficult task.

Some authors have associated the memory disturbances with the change in the brain waves revealed by electroencephalogram. Many observations indicate that there is a brain wave change, marked in the period after the convulsion. These brain waves return to normal in the matter of days or weeks. Hughes and his coworkers [14] state: "The longest period after treatment before the electroencephalogram returned to normal was three or four weeks."

2. Elation. Frequently, independent of or along with the amnesia, there is a period of elation. This cheery mood is striking, especially when compared with the previous profound depression. The most impressive fact about electro-coma treatment is this frequent change in mood. An individual who had been sad, droopy, and silent may, after several treatments, become active and happy. Occasionally the change does not stop at calmness, poise, and controlled activity. Indeed, some of these patients developed periods of marked overactivity resembling that of a hypomanic patient. There are effervescence, loudness, a stream of conversation, and extraordinary sociability. Such elation was shown by Mrs. T. V., whose case history was cited above. For several weeks after the series of electro-coma treatments, this formerly quiet and sad woman became talkative. She would address every patient on the ward, advising him or her to take electrical treatment. She would interrupt their eating, burst between two people who were talking to explain her views in a loud voice. There was a sparkling gleam in her eyes, and her face was wreathed in smiles. She walked about

the ward with a brisk step. She was overconfident of her ability, spoke optimistically of her plans. Gradually, during a period of several weeks the bubbles of effervescence burst and she became more calm. Two months after the treatment was over she had returned to her usual sense of well-being with a normal admixture of confidence and doubt, cheerfulness and sadness. It took about six weeks for the elation to subside.

3. Acute Confusional Episodes. A third type of psychologic reaction may be called an acute confusional syndrome. This state is often accompanied by marked amnesia. Patients lose the memory of their previous illness, but are neither calm nor elated. Instead they are tense, excited, hypersensitive to noises, and somewhat in a state of terror. They resemble patients with an acute toxic psychosis. Mrs. E. B. had been depressed for months and a course of treatment was started. After the fourth electro-coma reaction she became restless, sleepless, and developed marked fears. However, she did not discuss these fears openly. Instead, when the doctor approached her, she would pull away somewhat startled. She remained in a state of tension and alarm for 10 days. Such confusional episodes, a combination of bewilderment and terror, were more common in the older group than in the younger patients. Also, this side action was more frequent during the summer than in the spring or fall. Such disturbance is temporary and clears up after some days or weeks. When the confusion disappears, the patients usually return to a normal state. In not a single instance has this confusional period lasted more than several weeks.

Complications of Electro-Coma Therapy

A treatment as severe as electro-coma therapy has definite complications. In addition to the psychologic side actions, which are temporary, we must consider certain complications, possibly of a more permanent nature. Let us divide them into (1) immediate reactions and (2) possible later sequelae.

1. Immediate Reactions. The immediate complications include chiefly the injuries to the muscular and bony systems. With metrazol there was a fairly high incidence of fractures until patients were kept in hyperextension and restraint. This complication is much less frequent with the electro-coma method. Smith and his group [15] reported that the fracture incidence with the electrical method was one-fourth of that which occurred with metrazol. Furst and Stouffer [16] stated that there were some 3 per cent of fractures of the spine in a series of 115 patients. In our group we have had one bilateral fracture of the mandible and three cases of severe pain in the back. Two of these patients have shown fractures. The instances of fractured spine have occurred recently and for this reason we are planning to use intocostrin, as recommended by Bennett.[17]

2. Possible Late Sequelae. Inasmuch as the electric current traverses the frontal lobes of the brain, one must think of the possible late sequelae of this method. However, Cerletti and Kalinowsky believe that the brain

changes in man are reversible and do not produce serious damage. Further comment on the evaluation of such possible sequelae will be given later.

Other Studies

Along with the clinical studies reported, several research projects are being conducted. These will be mentioned briefly inasmuch as the papers, when completed, will be published elsewhere.

A. Electrocardiographic Studies. Dr. Edward Kline took a series of electrocardiographic records before and immediately after treatments in some 40 patients of this group. He has found even less change as a result of the electro-coma method than with metrazol,[18] with which therapy we encountered no significant cardiac disturbances. Although Dr. Kline's paper will cover the details, one may mention the fact that several of the patients in this group had known cardiac difficulties. One of the patients, Miss P. O., had once suffered from coronary disease and had been operated on by Dr. Claude Beck.[19] Two years later she developed a serious schizodepression which grew steadily worse despite all conservative therapy. As a last resort, after a consultation with Drs. Beck, Harold Feil, and O. P. Kimball, the family physician, a course of electro-coma treatment was given. This patient showed no circulatory difficulties as a result of 10 treatments and made a splendid recovery from her mental illness.

B. Anatomic Studies. Early this summer, in collaboration with Dr. E. Scharrer and Dr. D. Bodian, a Macacus rhesus monkey, whom we called Miss Darwin, was given a course of treatments. She received 10 treatments, on alternate days, in a series comparable with that of any private patient. The dosage was usually 200 milliamperes for 0.1 or 0.15 second. Each treatment consisted of a major reaction with coma and convulsion lasting from 30 to 90 seconds. (Ninety seconds was the longest duration of any convulsion obtained with the electrical method.) Miss Darwin withstood her treatment course very well and her behavior was in no way altered by it.

Four days after the last treatment the animal was placed in deep ether narcosis and perfused with saline and sodium nitrite, and then with Zenker's solution to which formalin had been added. The brain was thus hardened in situ by the intravascular perfusion to avoid the manifold artefacts which occur when the fresh brain is handled and sliced. Later the brain was removed, cut in cross section, and stained. Dr. Scharrer summarizes the findings as follows: "Sections from many regions of the brain revealed no lesions. Indeed, there were no cell destruction, no glial reactions, and no cellular changes in any of the sections."

This is the initial report of further work which has been planned.

C. Psychologic Studies. This summer Dr. Marguerite Hertz and Corinne Baker started a series of Rorschach tests taken before, during the course of treatment, and a month after the electro-coma treatment has been

concluded. The work is still in progress and the results of their findings will be published elsewhere.

D. Electroencephalographic Studies. A series of electroencephalographic tracings are being taken, parallel with the Rorschach tests, by Dr. Charles Henry of the Brush Foundation. These studies were begun only recently.

Comment

The most significant observation from the clinical standpoint is the fairly high rate of improvement as well as the willingness of patients to take this treatment. Few patients experience the dread so common with metrazol. Coercion is, as a rule, not necessary. Indeed, not a few of the patients were treated in a general hospital with their consent and coöperation.

In the face of the practical results obtained, one cannot but pause and consider the nature of the illnesses and the mechanism by which electro-coma therapy brings about some relief of the symptoms. Let us approach this interpretation by listing certain observations and deductions:

A course of electro-coma treatment influences the mood of the patient first. Quite uniformly in all types of patients, there is a tendency for the saddened patient to become more cheerful.

In patients who are troubled by serious hallucinations and delusions, the improvement in mood brings about a change in the patient's attitude toward his hallucinatory experiences. They are pushed into the background, dismissed, and ultimately lost.

Recovery in mental function occurs rarely as a result of one or two treatments, but requires the accumulated benefits accruing from a series of treatments.

Recovery often takes place even though the patient has gone through a period of mental clouding with marked forgetfulness or confusion. These psychologic side actions apparently do not interfere with recovery. The road to recovery appears to be obscured by the clouding, but despite this delay, the clouding lifts and the goal of recovery is reached.

The treatment achieves its success through direct electrical or chemical alteration in brain functions. This observation is self-evident, particularly in view of where the electrodes are placed, the unconsciousness, and the changes in the electroencephalogram.

The therapeutic effect is not attained through the mechanism of fear, as patients who receive such treatment do not emphasize any terror reaction as was common with metrazol.

The incidence of recovery is highest in depressed patients [20]; there is improvement in many cases of schizophrenia; and there is a tendency toward improvement in manic states.

During the course of electro-coma treatment, one observes quite regularly a change in direction of interest and thought. In most psychoses the

patient is introverted, his major attention is upon his viscera, his moods, his past. Even the hallucinations and the stimuli outward either arise from within or are interpreted from an egocentric viewpoint. During the course of treatment the patient tends to look outward, to notice external stimuli, and to interpret them in line with reality. Recovery is thus not a gain in total brain function as much as an improvement in the direction of thought.

On the basis of these observations, one may make several conjectures. The first is that the brain is the seat of or the major link of all types of psychoses. Psychoses are caused by disturbances in brain function and are believed to be organic, even if the disorder cannot be histologically established (Elvidge and Reed,[21] Katzenelbogen,[22] Hoskins,[23] Angyal, Freeman, and Hoskins [24]). The mechanism of recovery with electro-coma therapy depends upon an electrochemical change in brain cells. It appears as though each treatment damages the function of either cortical cells or association pathways. This effect tends to reduce or destroy those elements which are responsible for the abnormal direction of thought. The damage is reversible and there is a tendency for the abnormally functioning cells to regain their activity. Each succeeding treatment removes this tendency. In the depressions, when this change has been effectively brought about, there is recovery. In many types of schizophrenia, however, this improvement is temporary because there is an inherent destiny for a downward course. However, even in schizophrenia, so-called deterioration and dilapidation of thought are more apparent than real. One is sometimes surprised at the renewed intellectual interest in life which appears to be awakened in an apparently deteriorated patient as a result of shock therapy.

One must await the test of time before a true appraisal of the ultimate worth of the electro-coma method can be made. The permanence of recovery and the possibility of later complications will be determined only over a period of years. Scientific accuracy requires such a period. But the clinician who is faced with a sick patient must decide upon a course of treatment today. The practicing psychiatrist who examines a patient suffering from involutional melancholia, thin, trembly, agitated, and suicidal, cannot postpone action until an ultimate appraisal is made. He must use the best technic available at the time. At the moment electro-coma therapy appears to be the best available method for the treatment of many types of psychoses which have not responded to milder methods. The benefits to be gained and the dangers of inaction must be balanced against the hazards of the treatment. The advantages of electro-coma therapy over the hazards appear marked in involutional melancholia and many forms of depression. Likewise, the treatment is justified in many types of schizophrenia.

Conclusion

This paper records the clinical results in a series of 70 patients treated by the new electrical method. The term, "electro-coma therapy," is pro-

SUMMARY OF THERAPEUTIC RESULTS WITH ELECTRO-COMA THERAPY

No.	Name	Age	Diagnosis	Result
1	F. R.	49	Paranoid schizophrenia	Excellent
2	I. R.	29	Hebephrenic schizophrenia	Good
3	P. O.	55	Schizodepression	Excellent
4	D. P.	57	Agitated depression	Good
5	E. R.	65	Involutional melancholia	Excellent
6	B. L.	57	Endogenous depression	Excellent
7	S. M.	38	Endogenous depression	Excellent
8	W. B.	49	Agitated depression	Excellent
9	L. R.	25	Mania	Good
10	E. P.	23	Hebephrenic schizophrenia	Unchanged
11	S. B.	45	Endogenous depression	Excellent
12	B. I.	38	Paranoid schizophrenia	Doubtful
13	F. F.	36	Paranoid schizophrenia	Fair
14	I. H.	27	Paranoid schizophrenia	Unchanged
15	B. C.	67	Endogenous depression	Excellent
16	Z. L.	71	Involutional melancholia	Good
17	N. I.	54	Endogenous depression	Fair
18	B. H.	36	Paranoid schizophrenia	Good
19	C. E.	38	Endogenous depression	Good to fair
20	C. Ev.	47	Involutional melancholia	Good
21	F. M.	35	Hebephrenic schizophrenia	Temporary improvement
22	L. J.	19	Mania	Excellent
23	K. C.	46	Schizodepression	Fair
24	P. E.	30	Paranoid schizophrenia	Fair
25	D. N.	45	Schizodepression	Excellent
26	M. M.	29	Schizophrenia simplex (D. P.)	Fair
27	K. A.	34	Paranoid schizophrenia	Excellent
28	W. N.	55	Agitated depression	Fair (?)
29	M. H.	32	Schizodepression	Fair (?)
30	B. J.	61	Agitated depression	Fair
31	E. B.	45	Schizodepression	Good
32	T. V.	51	Agitated depression	Excellent
33	H. R.	30	Endogenous depression	Excellent
34	D. M.	57	Involutional melancholia	Good
35	F. J.	46	Paranoid schizophrenia	Poor
36	K. F.	37	Paranoid schizophrenia	Fair
37	W. C.	41	Endogenous depression	Excellent
38	F. M.	36	Compulsion neurosis	Unchanged
39	R. A.	60	Involutional melancholia	Poor (?)
40	S. M.	53	Paranoid schizophrenia	Unchanged
41	O. E.	34	Schizodepression	Fair
42	K. F.	55	Involutional melancholia	Good
43	B. I.	20	Paranoid schizophrenia	Unchanged
44	W. R.	19	Hebephrenic schizophrenia	Fair
45	W. G.	49	Schizodepression	Fair
46	L. K.	63	Agitated depression	Fair
47	M. B.	26	Schizophrenia simplex (D. P.)	Fair
48	M. D.	29	Endogenous depression	Excellent
49	B. S.	43	Agitated depression	Good
50	C. L.	50	Agitated depression	Excellent
51	P. H.	45	Endogenous depression	Good
52	U. M.	58	Agitated depression	Excellent
53	H. F.	52	Schizodepression	Good
54	P. M.	51	Schizodepression	Excellent
55	K. M.	35	Cyclothymia	Good
56	L. H.	51	Paranoid schizophrenia	Poor
57	H. H.	34	Endogenous depression	Fair
58	W. M.	45	Paranoid schizophrenia	Fair (?)
59	P. M.	24	Paranoid schizophrenia	Too early to judge
60	V. L.	62	Endogenous depression	Fair (?)
61	D. V.	45	Schizodepression	Good
62	S. F.	40	Agitated depression	Excellent
63	A. L.	46	Endogenous depression	Good
64	L. M.	38	Endogenous depression	Good
65	M. R.	59	Agitated depression	Excellent
66	S. B.	50	Schizodepression	Fair
67	M. M.	30	Schizophrenia simplex (D. P.)	Poor to fair
68	S. D.	31	Endogenous depression	Good
69	K. B.	50	Endogenous depression	Good
70	T. W.	62	Involutional melancholia	Good

posed in place of the label, "electro-shock therapy." "Minor," "moderate," and "major reactions" are used in place of the designations, "petit" and "grand mal."

The results of the electro-coma method have been found to be favorable. Certain of the psychologic side actions such as amnesia, periods of elation, and episodes of confusion are discussed. The complications are mentioned.

Certain general observations and a theoretical consideration of the mode of action of this method are offered.

For severe psychoses which do not yield to milder measures, electro-coma therapy is useful. It is particularly valuable for those psychoses in which a depressed mood change is a major factor. It is worthy of trial in many forms of schizophrenia.

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CHRONIC CONSTRICTIVE PERICARDITIS; A FOLLOW-UP STUDY OF THIRTY-SEVEN CASES *

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ALTHOUGH constrictive pericarditis has been recognized for many years, it was not until the advent of modern thoracic surgery and its meticulous attention to technic and anesthesia that surgical treatment of the disease has been possible, with gratifying results in small but important groups of cases, gathered especially in Boston,[1] Nashville,[2] and Cleveland.[3] An adequate historical review of the disease and its recognition has been given by White [1] and by Blalock and Burwell.[2] It is our purpose in this paper to present a follow-up study of 37 cases of constrictive pericarditis which have been seen

TABLE I

Group 1. Cases Cured by Operation

No.	Name	Sex	Age†	Onset	Seen†	Heart Size	Rhythm	Calcium	Etiology	Resection
1	C. S.*	F	19	1911	1925	normal	normal	0	?	7/18/28
2	B. K.*	F	10	1922	1931	+1 cm.	normal	+	?	11/16/33
3	L. F.*	F	30	1931	1933	normal	normal	0	?	4/6/33
4	C. F.*	M	19	1931	1933	normal	normal	0	pneumonia c pleurisy	4/22/33
5	L. C.*	M	12	1933	1933	normal	normal	+	?	7/12/33
6	O. P.	M	28	1935	1936	normal	normal	0	?	10/8/36
7	S. L.	M	26	1937	1938	normal	normal	0	?	6/29/38
8	H. W.	F	44	1937	1939	+1 cm.	normal	+	?	10/9/39

Addendum.

9. Case 4 of Group 5, I. B., really belongs also in Group 1, as does also
10. Case 2 of Group 2, G. P., and as will doubtless also, when adequate time elapses, several more.

11–14. Cases 5–8 of Group 2.

* Indicates cases reported by P. D. White in 1935.

† Age and year when first seen by us.

at the Massachusetts General Hospital, in whom the diagnosis has been made clinically and confirmed in all but a few by operation or by autopsy examination.

In 1928 Churchill carried out the first successful operation for this condition in this country,[4, 5] and in 1935 White [1] reported 14 other proved cases seen at the Massachusetts General Hospital. Twelve had been operated upon, three had not. Two more cases had been recognized at this hospital prior to the first operation but not confirmed at that time; both have died, and autopsy done on one of the two disclosed the presence of the disease.

Since that report in 1935, 20 additional cases have been studied, 16 of

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which have had pericardial resections (one of the 16 at another hospital), thus making a total of 37 patients seen at this hospital. Nineteen are living, 17 are dead, and one has not been examined lately and may be either living or dead. Twenty-eight of the group have been operated upon; nine have not.

Of the 37 patients, 25 were males, 12 were females. The ages ranged from 10 to 59 years. The average age of the entire group was 30.2 years, of those operated on 28.8 years, and of those not operated on 45.5 years.

Follow-up Analysis

For clinical evaluation of results, we have grouped the cases according to whether they (1) have been cured by operation, (2) have been improved by operation, (3) have died of the disease itself, (4) have died as a result of operation, (5) have died of complications, (6) are living without operation, or (7) have not been heard from in recent years.

*Group 1: Cured by Operation.** In this group at the present time there are eight cases, who were operated on from two to 13 years ago. These patients have all been seen or heard from in the past three months. In no case is there any limitation of activity because of a cardiac disability. They are able to carry on a normal life, and are in no way inconvenienced by the postoperative defect. Three of these patients show a just visible jugular pulse in the sitting position. Two patients were not examined, and although it is not known whether a venous pulse is visible at the present time, their general condition is excellent. Of the group reported in 1935 by White,[1] one patient (D. G.) who was thought to have been cured has since developed an enlarged heart, some slight edema of the legs, and an enlarged liver. However, she has no dyspnea, and is able to lead a fairly normal life, if exertion is not excessive. She is, therefore, included in the following group.

Perhaps the most instructive and interesting of all our cured cases is our very first patient (C. S., now C. S. O' N.) referred by one of us (P. D. W.) in the summer of 1928 to Dr. Churchill for pericardial resection. She was the first case successfully operated upon for this condition in this country. She has remained in excellent health ever since, was married a few years ago, and went through a normal pregnancy without any difficulty and delivery of a healthy son by forceps on March 1, 1941.

Group 2: Improved by Operation. In this group there are eight cases. One patient (D. G.) is mentioned above. A second patient (G. P.) has had two operations. Following the first operation he was greatly improved over his former condition, but continued to have some dyspnea and edema, and abdominal fluid collected which had to be removed by paracentesis every month or two. Two years after the first operation, further pericardial resection was carried out, and since then the patient has been able to carry on a

* Later note, August, 1942. Since this was written above, cases 5 to 8 of Table 2 have completely recovered, so that they plus case 4 of group 5 and case 2 of group 2, form an addition of 6 cases to Table 1, raising the cured cases from 8 to 14 in number.

TABLE II

Group 2. Cases Improved by Operation

No.	Name	Sex	Age	Onset	Seen	Heart Size	Rhythm	Calcium	Etiology	Resection	Remarks
1	D. G.*	F	11	1935	1935	+2 cm.	normal	0	?	2/27/35	Slight edema, enlarged heart and liver
2	G. P.	M	43	1934	1935	normal	aur. fib.	+	?	10/18/35 10/21/37	Slight dyspnea on exertion
3	P. C.	M	34	1931	1938	+2 cm.	aur. flut.	+	?	3/ 7/38	Last seen by us in 1938
4	W. R.	M	35	1936	1939	+2½ cm.	aur. fib.	+	?	2/ 9/40	Slight congestive failure, gallop rhythm
5	T. P.	M	24	1935	1940	normal	normal	0	?	11/12/40	
6	A. T.	F	40	1938	1941	+2½ cm.	normal	+	?	5/17/41	Too early to evaluate results completely.
7	R. C.	M	30	1935	1941	normal	aur. fib.	+	?	7/ 2/41	Probably cured
8	E. S.	F	24	1940	1941	normal	normal	0	TB	10/20/41	

* Case reported by P. D. White in 1935.

N.B. Later note, August, 1942. Cases 5 to 8 inclusive have shown complete recoveries and so should be transferred as "cures" from Table 2 to Table 1.

TABLE III

Group 3. Cases Who Died Primarily of the Disease Itself

No.	Name	Sex	Age	Onset	Seen	Heart Size	Rhythm	Calcium	Etiology	Treatment	Remarks
1	M. T.	M	59	1913	1914	+4 cm.	aur. fib.	0	?	diuretics belly taps	Died in congestion in 1922
2	A. A.	M	53	1924	1926	+1 cm.	aur. fib.	0	?	belly taps	Died in congestion in 1938
3	A. G.*	F	32	1933	1934	normal	normal	0	?	chest taps belly taps	Died in congestion in 1934 Large septal defect also found at autopsy
4	G. H.	M	25	1936	1937	normal	normal	+	?TB	Resection 3/22/37	Died of tuberculosis in 1937
5	M. S.	F	54	1938	1940	normal	aur. fib.	+	?	Resection 4/12/40	Died in congestion four months post-operatively
6	J. G.	M	45	1913	1938	normal	aur. fib.	+	?	diuretics belly taps	Died in congestion in 1940

* Case reported by P. D. White in 1935.

quiet life, though at times he is a little troubled by slight dyspnea on exertion. The venous pressure is now at the upper limits of normal. He has not required abdominal taps since the second operation, and may actually be considered a clinical cure. One patient (P. C.) who was successfully operated upon has not been heard from since four months after his pericardial resection three years ago, but at that time he reported that he was feeling quite well, that evidence of edema and ascites had practically disappeared, and that there was no dyspnea on exertion. It is probable that this patient represents a complete cure, but without further information it is deemed unwise to include him in Group 1. One patient (W. R.) operated upon in 1940 responded very well and was able to return to his work four months following operation. However, when seen two months ago, he showed some enlargement of his heart, chiefly in the region of the right ventricle, and there was a marked gallop rhythm over the right ventricle. This was believed to represent dilatation of the right ventricle following decortication. He was given digitalis, and improved somewhat, although the gallop is still present. In addition, he has developed an aortic diastolic murmur of unknown etiology since the operation. In spite of these sequelae, the patient seems able to carry on a moderately active life.

The remaining four patients of this group have all been operated upon within the last year, and we believe that it is too early as yet completely to evaluate their condition, but from the progress that each has made, we are under the impression that these remaining four cases will be included in Group 1 at a later date as cured cases.

One case (J. N.) reported as belonging to this group in 1935 by White [1] has since died, and will be discussed later. All this group of cases improved by operation has been seen since 1938.

Group 3: Cases Who Died Primarily of the Disease Itself. There are six patients included in this group, five having been added since 1935. Two of the three original cases (J. M., J. B.) died following operation and, therefore, are shifted to Group 4. Both showed definite evidence of tuberculosis of the pericardium. The third case (A. G.) was not operated upon, but died in acute heart failure before operation. At that time it was interesting to note that this patient showed a prolonged P-R interval (0.22 second) with prominent P-waves in Lead II, and is the only one in the entire series who showed this abnormality. One year later while the autopsy specimen was being mounted, it was discovered that there was a very large septal defect present in addition to the constricting pericarditis. This septal defect probably accounted for the unusual changes in the electrocardiogram, and perhaps for the very slight enlargement of the heart which was present.

Of the five additional patients, two were operated upon, and three were not. Of those subjected to surgery, the first (G. H.) did poorly after a stormy postoperative course, and after a second admission to the hospital two months later was discharged to a tuberculosis sanatorium where he subsequently died of tuberculosis. It was believed that operation on this patient

* Case reported by P. D. White in 1935.

stirred up activity in the mediastinal glands, although on section of the pericardium there was no evidence of old or recent tuberculosis, except for focal necrosis, which was not sufficient for a pathological diagnosis of tuberculosis. The second patient (M. S.) improved only slightly after operation, and during the succeeding four months required monthly abdominal paracenteses. She was readmitted to the hospital, suddenly went into acute heart failure, and died despite all efforts to save her. There was no evidence of tuberculosis at autopsy.

Of the three remaining patients, not operated upon, two were cases not discussed in the original report by White.[1] The first patient (M. R.) was originally seen in 1913, when diagnoses of hepatic cirrhosis, ascites, and cardiac hypertrophy were made. This patient was followed for nine years, during which time the diagnosis was changed to chronic constrictive pericarditis. During the nine years of observation the patient had repeated abdominal paracenteses, and on his last entry died very shortly after admission in acute and chronic congestion. No autopsy was performed. The second patient (A. A.) was followed for 14 years, but because of his age and only moderate disability, operation was not advised even though the condition later became more advanced. He had many abdominal paracenteses and died in 1938 in congestion. Autopsy confirmed the clinical diagnosis. The third patient (J. G.), seen since our original series was published, was first examined in 1938, at which time operation was not considered because the condition was mild. He was seen the following year, during which time he had a marked progression of the disease. He was then admitted in preparation for operation. However, he did not respond sufficiently well to treatment to warrant the operative procedure, and was discharged for further care, to return for operation at a future date. However, he failed to make the expected response and died in congestion. Autopsy confirmed the clinical diagnosis.

Group 4: Cases Who Died as a Result of Operation. In this group there are five cases, three of which (J. B., J. H., J. M.) were included in the 1935 report in Groups 3 and 4, but have been placed in the present Group 4, because their deaths were due essentially to the surgical procedure. Of the two additional cases, one (S. G.) died, on the operating table, of acute dilatation and failure of the right ventricle when the pericardium was removed. The second case (V. G.), although seen here, was operated upon at another clinic and died two days postoperatively following pulmonary embolism. It was of interest that in this case there was a core of calcification which extended from the pericardium through the myocardium to the endocardium at one point. These five deaths in the series of 28 cases submitted to operation result in an operative death rate of 18 per cent.

Group 5: Cases Who Died of Complications. In this group are six cases, three of which were reported in 1935. The third case (J. H.) originally in this group has now been included in Group 4. The third case in the present group is that originally reported as improved by operation (J. N.). In

TABLE IV

TABLE IV

Group 4. Cases Who Died as a Result of Operation

No.	Name	Sex	Age	Onset	Seen	Heart Size	Rhythm	Calcium	Etiology	Resection	Remarks
1	J. B.*	M	23	1931	1931	+3½ cm.	normal	0	TB	1/ 4/31	Died one day postoperatively
2	J. H.*	M	52	1931	1933	+2 cm.	aur. fib.	+	?	9/20/33	Died of bronchopneumonia, pulmonary edema six days postoperatively
3	J. M.*	M	36	1934	1934	+4 cm.	normal	0	TB	4/ 2/34	Died on operating table
4	S. G.	F	13	1934	1936	normal	normal	0	?	5/27/36	Died on operating table, acute right ventricular dilatation and failure
5	V. G.†	M	41	1929	1938	normal	normal	+	?	8/20/38	Died of pulmonary embolism two days postoperatively

* Cases reported by P. D. White in 1935.

† Operated on at another clinic.

TABLE V

Group 5. Cases Who Died of Complications

No.	Name	Sex	Age	Onset	Seen	Heart Size	Rhythm	Calcium	Etiology	Treatment	Remarks
1	L. A.*	M	18	1930	1930	+3 cm.	normal	0	TB	Resections 4/16/31 2/23/34	Died of acute pancreatitis one month after second operation
2	H. P.*	F	31	1928	1931	+1 cm.	normal	+	sepsis	Resection 4/29/31	Died of general sepsis one year postoperatively
3	J. N.*	M	16	1921	1932	+2 cm.	aur. fib.	+	with pleurisy pneumonia	Resections 9/27/32 11/16/33 Omentopexy 3/30/34	Died suddenly one day after removal of emboli of common iliac arteries and right femoral artery, 1940
4	I. B.	M	47	1935	1935	normal	normal	0	?	Resection 11/ 6/35	Apparent cure. Died of type III pneumococcus pleurisy one year postoperatively
5	M. B.	F	34	1928	1938	+2 cm.	aur. fib.	+	?	Resection 5/16/38	Died of bronchopneumonia two years postoperatively
6	W. M.	M	33	1934	1936	normal	normal	0	TB	Diuretics	Died of miliary tuberculosis, 1936

* Cases reported by P. D. White in 1935.

this latter case, two pericardial resections and an omentopexy had been performed, but the patient continued to have large accumulations of fluid in the abdominal cavity, requiring frequent paracenteses. He was able, however, to carry on moderate activity, as long as the abdominal fluid was removed at frequent intervals. When retiring one evening he suddenly experienced numbness and coldness of the lower extremities. A diagnosis of peripheral emboli of the legs was made, and emboli were removed from the left and right common iliac arteries, and from the right femoral artery. One day following the operations he suddenly began to gasp for breath and died in a few minutes. The autopsy protocol describing the heart, lungs, and mediastinum is as follows:

An area (measuring approximately 6 by 5 cm.) of the lower anterior portion of the pericardium had been surgically removed. In its place is a moderate amount of fibrous scar tissue which is adherent to the under surface of the operative wound, but which separates easily with blunt dissection. The remaining portion of the anterior parietal pericardium is for the most part fibrous and adherent to the visceral layer. The upper two-thirds of the left antero-lateral portion of the pericardial cavity is completely obliterated by irregular confluent masses of calcium, extending cephalad to involve the base of the aortic valve and forming a large plaque measuring 9 by 6 cm. in extent. The outer surface of the plaque is covered by fibrous tissue which in turn is adherent to the mediastinal parietal pleura of the left lung. This plaque extends somewhat posteriorly to impinge by external pressure upon the lumen of the mitral valve. There is no calcification of the pericardium in the region of the apex of the left ventricle. There is a similar but smaller zone of pericardial calcification involving the anterolateral aspect of the right ventricle over an area measuring 9 by 4 cm. On this side, however, the calcification extends almost to the apex of the right ventricle, but does not extend up as high as the level of the pulmonary artery. Posteriorly, the pericardium is free from calcification.

Heart. The heart is not dissected free from the pericardium and the lungs, but is opened in situ. The myocardium is red-brown. The right ventricular wall measures 3 to 4 mm. in thickness, the left, 13 to 15 mm. The left auricle is moderately enlarged, and measures roughly 8 by 6 by 4 cm. The remaining cavities and the columnae carneae are essentially negative. The lateral aspect of the mitral valve is pressed on externally by the calcified pericardium, resulting in a slight stenosis. The aortic valve is moderately thickened and shows slight interadherence of the cusps at their commissures. The other valves are essentially negative. The coronaries are negative.

There was no evidence of a pulmonary embolus of such a size that death could be attributed to it.

It is interesting that in this case there was almost complete constriction of the left ventricle together with an impingement by a calcified pericardial plaque on the lumen of the mitral valve from outside the heart, which factors undoubtedly accounted for much of the lack of improvement following pericardial resections and omentopexy. This situation had been suspected before death. It is also of interest that chronic rheumatic endocarditis with slight aortic stenosis was present. This is the only case in the entire series in which rheumatic heart disease has been found. There was no pulmonary embolus of sufficient size to account for the sudden death.

The fourth case (I. B.) had apparently been cured when seen one year following operation. He had been able to carry on a fairly normal life until about five days before his last hospital admission when he suddenly developed a respiratory infection followed by dyspnea and cyanosis. In spite of vigorous treatment he died. Postmortem examination showed pyopneumothorax, type three pneumococcus, and pulmonary congestion, but no frank consolidation.

The fifth case (M. B.) underwent pericardial resection in 1938, and was seen again in 1939, when it was noted that she had very little, if any, improvement from operation, and it was advised at that time that if there were no definite improvement in the next few months another operation should be attempted in the hope of further improving her condition. However, the patient did not return for further study, and she died elsewhere of bronchopneumonia. An autopsy showed, in addition to the constrictive pericarditis, healed obliterative pleuritis, healed fibrous peritonitis, a tubercle in one lung, apparently healing, and cardiac hypertrophy.

The sixth patient (W. M.) was first seen by us in 1936, after a pericardial tap had revealed slightly bloody fluid with tubercle bacilli demonstrated in the smear. The patient was advised to have a period of prolonged bed rest, both for treatment of the active tuberculosis, and in the hope that he could be adequately prepared for operation after subsidence of the active infection. However, the patient failed to improve, and died four months later. Postmortem examination showed miliary tuberculosis.

Group 6: Patients Living, Not Proved by Operation. There are three patients in this group, two of whom have been seen since the original report of cases from this hospital. The first patient (W. B.) included in the original reports, and now 68 years old, has written that he is feeling well, with only slight restriction of activity. He is continuing with his medical practice, "feeling better than 10 years ago." There were some slight hepatic congestion and engorgement of the neck veins. He has shown pericardial calcification by roentgen-ray. His most recent letter is of such great interest and importance in showing that operation is not essential in every case that it is herewith quoted in full:

> Your letter of inquiry received. Am glad to tell you I am doing very well indeed.
>
> *Exercise tolerance:* Well, I think I can get around just as well now as I did 10 years ago. Can handle one flight of stairs very well and two flights also without discomfort. Outside, I don't walk more than a block. Don't choose to walk but can do so if put to it.
>
> *Edema:* During the last nine months have had no edema at all anywhere, at any time of the day or night.
>
> *Irregular heart action:* My heart is still irregular but does not bother me as it did except when I hurry, get excited or overdo in any way, so I am careful. In fact, I think in the morning, after a good night's rest and sleep, my heart is regular and can go for several days without being conscious of a flutter at all.
>
> *Liver enlargement:* Have forgotten about the old liver now. For the last half-century or a little less, that gave me a lot of distress, enlarging and contracting with

varying degrees of cardiac weakness from an inch or so below the costal border down to the umbilicus. Don't believe liver is enlarged at all now. Have no sensation of any fullness in that region now.

Engorgement of veins of neck, particularly when in the recumbent posture: That always gave me a lot of distress down through the years. It was my habit to stuff a corner of the pillow firmly under my jaw under-side to relieve that discomfort in order to get sleep. Don't have to do that now. So, doctor, that is how I am at the present time. People tell me I look as young as I did 20 years ago. Have had no setbacks recently of infections in the past eight or ten years. That means a lot. Don't think I could stand much of an infection. Nine months ago, in February of this year, had a coryza. That crept down to the bronchi and gave me a troublesome cough. I felt quite sick. No temperature. Had some edema then in face and over sacrum and in ankles when I got on my feet. That was nine months ago.

I think I had also some crepitations at base of lungs. Sent for my doctor. He said, "Get right to the hospital to bed and stay there for four weeks. Absolute rest in bed, no getting up for anything. Getting up once will set you back one week at $5.00 per day." So that was a pretty good incentive to obey orders. For the first week I slept all night and dozed most of the days between feedings. After three weeks, I felt so good I kicked over the traces, got up, sat around for one day, and went home the next. My legs were weak from non-use, but all the edema had vanished and I haven't had any since, and that's a record for me, and since then, have been, as I told you above.

A recapitulation of the whole condition for the past 50 years: In the fall of 1893, went to Baltimore to resume medical studies. In the winter of 1894 had a subacute pleurisy. Continued my studies and attended class, holding on to my right side. Saw Dr. Latimer, a prominent physician in Baltimore then, regarding this. He examined me and prescribed Beechwood Creosote. Evidently thought I had tuberculosis. Returned home to Nova Scotia after college closed.

I then became severely ill with an acute febrile condition. In bed three months. After getting up, had a lot of dropsy and a large pulsating liver. Was treated for typhoid fever. In the fall of 1894, went to Boston and consulted Dr. Frederick Shattuck. Was in Massachusetts General Hospital one month, the month of November, 1894, under Dr. Shattuck's care. Had a lot of swelling then and enlargement of the liver. Dr. Shattuck's diagnosis was chronic hepatitis due to extension of the pleurisy through the diaphragm into the liver. In the fall of '95 returned to Baltimore to resume medical studies. I still had all that winter a lot of swelling in my legs. Graduated in medicine in 1896. I returned to Nova Scotia and did a hard country practice for 18 years. Continued getting better as the years went by but at no time was I free from edema of the legs.

In 1914, the beginning of the first year of war, I enlisted in the army. That was too strenuous for me. In the winter of 1917, had an acute heart failure, in bed one year. Gradually pulled out of that. In 1923, began having attacks of auricular fibrillation. In 1929, another attack of heart failure. In bed one year. About that time I first saw you, and you know how I have been since then.

When you next want to know how I am getting on, I can only hope that I will be here to answer you.

The second patient (C. A.) was seen one year ago, having had some slight edema, and swelling of his abdomen from time to time during the previous four years. However, his condition one year ago was not considered serious enough to warrant operation, and he was discharged from the hospital. He was seen by us again one year later, and was doing well on a

TABLE VI

Group 6. Cases Living, Not Proved by Operation

No.	Name	Sex	Age	Onset	Seen	Heart Size	Rhythm	Calcium	Etiology	Treatment	Remarks
1	W. B.*	M	57	1917	1930	+1 cm.	aur. fib.	+	?	Diuretics Digitalis	Slight restriction of activities
2	C. A.	M	50	1936	1940	+1 cm.	aur. fib.	+	?	Diuretics Digitalis	Feeling well. Slight limitation of activities
3	A. J.	M	36	1921	1941	+1 cm.	aur. fib.	0	?	Diuretics	Moderate limitation of activities

Group 7. Case Not Examined Recently

No.	Name	Sex	Age	Onset	Seen	Heart Size	Rhythm	Calcium	Etiology	Treatment	Remarks
1	A. B.*	M	45	1932	1932	+1½ cm.	aur. fib.	+	?	Diuretics Digitalis	Last seen in 1934

* Cases reported by P. D. White in 1935.

maintenance dosage of digitalis, and quinidine for occasional attacks of palpitation. Only occasionally was there any edema, and the patient thought he was feeling better than he had previously felt, although occasional rest periods were found helpful. Operation was not advised.

The third patient (A. J.) had had a story of about 20 years of slight edema and shortness of breath which had become somewhat troublesome in the past year, due to tachycardia incident to the onset of auricular fibrillation. Under digitalis therapy his heart rate fell, and he improved and was able to continue with his work with some limitation. It was thought that there was not enough disability to warrant the risk of operation at the present time. This patient was seen only a few months ago for the first time.

Group 7: Case Not Recently Examined. There is one patient (A. B.) in this group who has not been heard from since the original report; efforts to locate him have failed. This patient was not operated upon.

Etiology

Among our original 15 cases which were reported in 1935, the etiological factors were tuberculosis in three cases, pneumonia with polyserositis in two, sepsis in one, and unknown or questionable in nine. Among the additional 22 cases, the etiological factors were tuberculosis in two, and unknown or questionable in 20; five of this latter group had had a definite history of pleurisy (three cases) or pericarditis (two cases). It is of interest that one of the cases without a known etiological factor (J. G.) had suffered from an attack of rheumatic fever with pancarditis 25 years before he was first seen by us, and had had several bouts of rheumatic fever since the first infection, but at autopsy there was no evidence of any rheumatic valvular disease or shortening of the chordae tendineae. It seems unlikely that rheumatic fever played any important part in the production of the constricting pericardium, since almost certainly some damage to the heart valves or the chordae tendineae would be expected if such severe damage of the pericardium were caused by rheumatism. One other case (J. N.) showed rheumatic endocarditis with slight aortic stenosis; it is possible that rheumatism may have been the etiological factor in this case, but it is more likely that the rheumatic heart disease was a coincidental finding and that an attack of pneumonia was the underlying cause. One other case (M. B.) had had an attack of rheumatic fever 10 years prior to operation, but at autopsy there was no evidence of any rheumatic heart disease. These results are in accord with an autopsy study of 6100 cases at the Massachusetts General Hospital, reported by Sprague, Burch, and White,[6] in which there were only six cases of adherent pericardium without valvular disease who gave a history of rheumatic fever, and in none of whom Pick's disease was present. Also a follow-up study of 1500 cases of rheumatic fever at the House of the Good Samaritan in Boston has revealed no case of chronic constrictive pericarditis (personal communication, E. F. Bland, 1941). Although several authors have mentioned rheumatic fever as being an etiological factor in the pro-

duction of constrictive pericarditis (Trout, Smith and Willius, and Rothstein, cited by Blalock and Burwell [2]), we are quite sure that rheumatic fever is rarely, if ever, an etiological factor.

One case (A. G.), cited above in Group 3, had, in addition to the constrictive pericarditis, a congenital defect, consisting of a large patency of the septum.

Tuberculosis appears as a less common factor in our series than in that of Blalock and Burwell, accounting definitely for only 14 per cent of our cases (5 of 37), whereas it accounted for 64 per cent of theirs.

Size of Heart Area

In this study the size of the cardiac area has been of interest. Of the total number of 37 cases, 19 had slightly to moderately enlarged areas, i.e., from 1 to 4 cm. over normal in transverse diameter during life, and 18 had normal sized hearts. The measurements were made by roentgen-ray or physical examination, or both. In addition, some of the cases (10 in all) showed murmurs, systolic in all except one instance (see table). Calcification of the pericardium was encountered in 16 cases. These findings are tabulated below:

Table VII

Incidence of Heart Area Enlargement, of Murmurs and of Pericardial Calcification in Our Total Series of 37 Cases

	Number of Cases	Murmurs	Calcification
1. Enlarged heart areas:			
Living, operated on	6	1-apical systolic 1-aortic diastolic	5
Living, not operated on	3	0	2
Dead, operated on	7	1-apical, aortic systolic 1-apical systolic	3
Dead, not operated on	2	2-apical systolic	0
? Living or dead	1	0	1
Total	19	6	11
2. Normal sized hearts:			
Living, operated on	10	2-apical systolic	3
Living, not operated on	0	—	—
Dead, operated on	5	1-apical systolic	1
Dead, not operated on	3	1-apical systolic	1
Total	18	4	5

From this it can be seen that slightly over half of the patients had enlarged heart areas. Forty-three per cent of the cases had calcification of the pericardium, but of those with normal sized hearts only five (28 per cent) had calcification, whereas of those with enlarged heart areas, 11 (60 per cent) had calcification. The difference in the presence or absence of murmurs in the two groups is not striking. The one aortic diastolic murmur developed one year following operation. None of the apical systolic murmurs were of marked intensity. From the figures presented above it would

appear that those patients with normal sized hearts have a somewhat better prognosis than those with enlarged heart areas. Those with calcification are more likely to show enlargement.

Electrocardiographic Findings

The electrocardiograms were studied in these cases with respect to rhythm, axis deviation, voltage changes, and T-wave changes.

1. Rhythm. The arrhythmias in the various groups are tabulated, together with heart size and calcification:

Table VIII
Incidence of Arrhythmias

	Enlarged Heart Area	Calcification	Normal Heart	Calcification
1. Normal rhythm:				
Living, operated on 12	4	3	8	1
Living, not operated on 0	—	—	—	—
Dead, operated on 8	5	1	3	1
Dead, not operated on 2	0	0	2	0
22	9	4	13	2
2. Auricular fibrillation:				
Living, operated on 3	1	1	2	2
Living, not operated on 3	3	2	0	0
Dead, operated on 4	3	2	1	0
Dead, not operated on 3	1	0	2	1
? Living or dead 1	1	1	0	0
14	9	6	5	3
3. Auricular flutter:				
Living, operated on 1	1	1	0	0

From the above it can be seen that normal rhythm is more common than auricular fibrillation or flutter, and was present in two and a half times as many normal sized hearts as was fibrillation or flutter. However, the presence of an arrhythmia was noted two and a half times as frequently in the enlarged hearts as in the normal sized hearts. Also, abnormal rhythms were present three times more frequently in cases which showed calcification than in those which did not.

The patient (P. C.) with auricular flutter continued to have this arrhythmia for three weeks after operation, which was the time of the last tracing recorded.

One patient (A. T.) showed transient auricular flutter a few days postoperatively, but normal rhythm returned following the administration of quinidine, and has remained in the subsequent six months.

One patient (C. A.) when first seen showed a shifting pacemaker, but when seen one year later showed auricular fibrillation. He had not been operated upon.

One other patient (J. N.) developed auricular fibrillation one year after the first operation. The arrhythmia persisted until his death.

2. Axis Deviation. Although axis deviation in the presence of the low voltage found in most of the electrocardiograms is not of great importance, it was found that one patient (L. F.) shifted from a slight right axis deviation to normal eight and a half years after operation, without much change in voltage; five cases shifted from normal to slight right axis deviation (O. P., G. H., J. N., I. B., M. B.) three months to five years following operation. Two of these (J. N. and M. B.) showed auricular fibrillation. One patient (M. T.), not operated upon, shifted from left axis deviation to a 90 degree angle over a period of five years. He also showed right bundle branch block, and was the only patient of the 37 with widened QRS complexes. In none of the cases, however, were these changes striking.

Follow-up electrocardiograms were obtained on 30 of the 37 patients, 22 who had been operated on, and eight who had not. For purposes of discussion, the QRS complex changes and the T-wave changes of the classical leads will be discussed separately.

3. Low Voltage. Low voltage of the QRS complexes (and T-waves) was found in all three classical leads in 18 of the 28 cases operated upon. Following operation, a slight increase in voltage, averaging 2 mm., was found in two cases (B. K., R. C.). Practically no change was found in 12 cases. The remaining four cases (C. S., L. A., J. H., J. N.) showed lower voltage after operation. These figures indicate that there is neither a marked nor consistent change in the voltage of the QRS complex following pericardial resection, and that the voltage usually remains low after operation.

Of the eight cases not operated upon, low voltage was present in all three leads in four. When the eight cases were seen later, four had remained practically the same (two months to two years after the original electrocardiogram), and four showed lower voltage throughout (from one to 13 years after the original electrocardiogram).

4. T-Waves. The T-wave changes in Leads I and II, before and after operation, are best shown by the following table:

TABLE IX

The T-Waves in the Electrocardiograms of Cases Operated Upon

Before Operation		After Operation					
	Number of Cases	Normal	Low	Flat	Diphasic	Inverted	Unchanged
Normal T_1	1	0	1	0	0	0	0
Low T_1	12	1	0	2	2	3	4
Flat T_1	1	0	0	0	1	0	0
Diphasic T_1	1	0	0	1	0	0	0
Inverted T_1	7	0	1	1	0	0	5
Normal T_2	1	0	0	0	1	0	0
Low T_2	5	0	0	3	0	1	1
Flat T_2	0	—	—	—	—	—	—
Diphasic T_2	6	0	1	1	0	3	1
Inverted T_2	10	0	0	1	2	0	7

Of the above 22 cases in which follow-up electrocardiograms were obtained, six had both T_1 and T_2 inverted. Of these, four showed inverted T-waves in Leads I and II after operation. Of the remaining two, T_1 remained inverted in both cases, T_2 became diphasic in one case and flat in the other.

The one case in which a flat T_1 became normal after operation was one of those in whom the voltage also became normal (L. C.). Of the 22 cases, in six T_1 became higher (including that in which T_1 returned to normal), and in 16 cases T_1 remained unchanged or became lower. From these results it would seem apparent that there is no consistent or characteristic change of the T-wave in Lead I or II following pericardial resection.

Of the eight cases not operated upon in which follow-up electrocardiograms were obtained, seven showed the following changes in the T-waves of Leads I and II:

TABLE X

T-Waves of Electrocardiograms of Cases Not Operated Upon

Original Tracing		Follow-up Tracing						Time Interval
	Number of Cases	Normal	Low	Flat	Diphasic	Inverted	Unchanged	
Low T_1	4	—	—	1	1	1	1	1 mo. to 13 yrs.
Flat T_1	2	1	1	—	—	—	—	2 mo. to 2 yrs.
Diphasic T_1	0	—	—	—	—	—	—	—
Inverted T_1	1	—	—	1	—	—	—	9 mo.
Low T_2	2	1	—	—	1	—	—	2 mo. to 1 yr.
Flat T_2	1	—	—	—	—	—	1	2 yrs.
Diphasic T_2	3	—	—	1	—	1	1	1 to 13 yrs.
Inverted T_2	1	—	—	—	—	—	1	9 mo.

These figures show that in four cases there was some slight increase of the T-waves during the period of observation of the cases not operated on. The changes do not appear consistent enough to warrant any conclusion regarding the change in T-waves of Leads I and II during the progression of the disease.

The eighth patient showed right bundle branch block in all tracings, with upright T_1 and inverted T_2 in the first tracing. The last tracing, five years later, showed a marked decrease in voltage, and T_1 had become inverted and T_2 had become upright. The T-wave changes cannot be interpreted accurately in this case with reference to the constricting pericarditis, because of the complicating right bundle branch block.

In three cases, T_3 became normal after operation (O. P., A. T., E. S.). In one case (A. G.), not operated upon, T_3 became normal two months after the original tracing. In all other cases T_3 remained flat or inverted after either operation or observation.

We would emphasize herewith the importance of the electrocardiogram in the diagnosis of chronic constrictive pericarditis; it was abnormal in all

our cases, the abnormality consisting of low voltage of the QRS waves or abnormal T-waves, or both.

Summary

Thirty-seven cases of constrictive pericarditis are presented as a follow-up study. The later results of 15 of the cases which were reported in 1935 are given, together with a brief clinical survey of the course of 22 additional patients seen at the Massachusetts General Hospital. The age, sex, etiological factors, clinical data, and electrocardiographic findings are presented:

(*1*) *Age.* The ages ranged from 10 to 59 years. The average age of those operated upon was 28.8 years, of those not operated upon 45.5 years.

(*2*) *Sex.* Male, 25; female, 12.

(*3*) *Etiology.* Tuberculosis, five; sepsis, one; pneumonia with polyserositis, two; unknown or questionable, 29. Of this latter group three cases had had known previous pleurisy, and two had had known acute pericarditis.

(*4*) *Heart size.* Nineteen, or 54 per cent, of the patients reported had enlarged heart areas, from 1 to 4 cm. over normal in tranverse diameter. Of these, five or 26 per cent, had apical systolic murmurs; one had an aortic diastolic murmur. Eighteen, or 46 per cent, had normal sized hearts. Of these, four, or 22 per cent, had apical systolic murmurs.

(*5*) *Calcification of the pericardium.* Sixteen, or 43 per cent of the total number of cases in this series, had calcification of the pericardium. Eleven, or 30 per cent, had enlarged heart areas with the calcification of the pericardium, and five, or 13 per cent, had normal sized hearts with calcification of the pericardium.

(*6*) *Electrocardiographic Findings.*

(*a*) Normal rhythm was present in 22, or 60 per cent, of the series. Auricular fibrillation was present in 14, or 37 per cent, of the series. Auricular flutter was present in one, or 3 per cent, of the series. Normal rhythm was found to be present two and a half times more often than was auricular fibrillation or flutter in the cases with normal sized hearts. Arrhythmia was present two and a half times as frequently in the case of the enlarged heart areas as in the case of the normal sized hearts.

(*b*) Axis deviation was found to be normal in almost all of the patients.

(*c*) Low voltage of the QRS waves in all three classical leads was found in 22 cases, or 60 per cent, of the whole series. Following operation, normal voltage of the three leads was found in two, or 7 per cent, and these two cases had had normal voltage of the QRS waves in two of the three classical leads before operation.

(*d*) Abnormal T-waves of two or three of the classical leads were found in all cases in this series. Following operation none of the T-waves became

normal in more than one lead; three cases showed normal T-waves in Lead III, and only one case showed normal T-waves in Lead I.

(*e*) The electrocardiogram was abnormal in every case.

(*7*) *Follow-up Status* (August, 1942). Twenty-eight of the 37 patients have had pericardial resection.

(*a*) Fourteen of these 28 were clinically cured although in a few cases slight increase in venous pressure above the average normal remains, and one of the 14 died of other cause (pneumococcus pleurisy) one year post-operatively.

(*b*) Three others were much improved by the operation.

(*c*) Two died primarily of the disease itself, one of tuberculosis soon after the operation and one in congestion four months post-operatively.

(*d*) In five the death was related to the operation, 2 of the 5 succumbing on the day of operation, one died one day post-operatively, one died of pulmonary embolism two days post-operatively, and one died of bronchopneumonia six days post-operatively.

(*e*) Four died of other complications: one of acute pancreatitis one month after the second operation, one of general sepsis one year post-operatively, one of bronchopneumonia two years post-operatively, and one of arterial embolism seven years post-operatively.

(*8*) Nine cases did not have pericardial resection.

(*a*) Three were so mild that operation was not thought necessary.

(*b*) Three were too sick to undergo operation.

(*c*) One died of miliary tuberculosis before the operation could be carried out.

(*d*) One died before this series of operations was started.

(*e*) One case has been lost sight of.

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SURGICAL TREATMENT OF HYPERTENSION (RESULTS IN FIFTY-FOUR CASES)*

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DURING the last 10 years 54 patients with hypertension have been operated upon at the Lahey Clinic. Sufficient time has elapsed to justify an evaluation of the results to date in all of these cases. The effectiveness of surgery in some cases of hypertension has been reported by Allen and Adson,[1] Crile,[2] Peet [3] and others. As we cannot predict with certainty the ultimate outcome of surgery in individual cases, a more accurate method of selection remains to be discovered.

The first patient was operated upon in 1931. Up to the early part of 1935, fourteen patients were submitted to operation (Group A, table 1). Group B includes those patients operated upon since the second half of 1935 and were more carefully selected. An average of 10 patients per year in Group B have had operations. We have limited our selection to patients with Grades 2 and 3 hypertension,† but with the additional requirement that they be under 40 years of age, and that there must be no evidence, or at best, slight evidence of widespread vascular disease as determined by a study of the heart, kidneys and optic disks. Furthermore, the blood pressure must fall to 150 mm. systolic and 100 mm. diastolic while the patient is at rest or under drug sedation. A few exceptions were made, however, especially in the matter of age, as indicated in table 1.

The first 13 patients (Group A) were subjected to a supradiaphragmatic splanchnicectomy and ganglioramisectomy, and the remainder to a two stage subdiaphragmatic or transdiaphragmatic resection of the greater and less splanchnic nerves, with the removal of the twelfth thoracic, first and second lumbar ganglia, the visualization of the adrenal glands and biopsy of each kidney. If a sufficient amount of omentum were available, the left kidney was decapsulated and a nephro-omentopexy carried out. If large accessory arteries penetrated the capsule of the kidney or the omentum was bound down by previous operative procedures, the nephropexy was not attempted.

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† Grading according to Keith, Wagener and Barker.[4]

Grade 1—Mild hypertension which is normal at bed rest. Slight sclerosis of retinal vessels.

Grade 2—Moderate, fluctuant hypertension which remains above normal at rest and under sedation. Moderate sclerosis of retinal vessels.

Grade 3—Moderate to severe hypertension with little fluctuation. Sclerosis of retinal vessels with angiospastic retinitis.

Grade 4—Persistently high blood pressure. Marked sclerosis of retinal vessels with diffuse retinitis and edema of optic disks.

TABLE I

Results of Surgical Approach for Hypertension

Year		Number of Patients	Grade of Hypertension			Average Age	Operative Deaths	Postoperative				
								Deaths		Blood Pressure Change		
			II	III	IV			No. of Cases	Average Duration	None	Slight	Satisfactory
1931	A	1	1	—	—	51	—	—	—	1	—	—
1934	A	3	1	1	1	46	—	1	6 mo.	3	—	—
								1	4 yr.			
1935	A	9	2	2	5	41	—	7	20 mo.	9	—	—
	A	2	2	1	—	31	1	—	—	—	—	1
1936	A	1	1	—	—	31	—	—	—	—	1	—
1937	A	9	8	1	—	35	—	0	—	3	3	3
1938	B	7	6	1	—	36	—	1	2 mo.	4	2	1
1939	A	10	7	3	—	39 5 over 40	—	—	—	6	2	2
1940	A	12	11	1	—	34 1 over 40	1	—	—	2	3	6
Total		54	39	9	6		2	10		28	11	13

TABLE II

Blood Pressure Changes in Patients Recorded as Having a Good Response

Case	Before Operation *		After Operation †		Time since Operation
	Blood Pressure, mm.		Blood Pressure, mm.		
	Systolic	Diastolic	Systolic	Diastolic	
1	228	120	140	80	8 months
2	236	140	150	108	1½ years
3	190	122	146	96	3½ years
4	200	130	140	94	2½ years
5	258	140	150	100	2½ years
6	208	120	148	90	2½ years
7	220	140	140	100	2 years
8	174	110	144	90	1½ years
9	200	110	140	90	9 months
10	224	132	120	82	1 year
11	190	120	152	94	13 months
12	180	130	136	110	5 years
13	206	140	154	100	7 months
	Systolic	Diastolic	Systolic	Diastolic	
Range	174–258	110–140	120–154	80–110	
Average	208	126	144	94	23 months

* Average of two or more readings taken at the clinic during initial examination after patient had been in recumbent position for 15 minutes.

† Average of two or more monthly readings taken at the clinic after patient had been in recumbent position for 15 minutes.

Two postoperative deaths (3.6 per cent) occurred. One patient died two weeks after leaving the hospital from a pulmonary embolism secondary to thrombophlebitis. The second patient had a cerebral hemorrhage 12 days after operation. Except for these two deaths the postoperative course of all the patients was without serious incident, the usual hospital stay being approximately three weeks.

We have been fortunate in being able to follow the course of all of these patients since their operation. Nine of the 13 patients in Group A seen prior to the middle of 1935 have died. Death occurred an average of 20 months after operation. One patient died four years after operation. The causes of death were related to hypertension, death being cerebral, cardiac or renal in nature. Four patients in this group are still alive at the time of writing this paper, the blood pressure remaining elevated as before operation.

There are 41 patients in Group B. Thirty-five had Grade 2 and six, Grade 3 hypertension. All but five were younger than those in Group A. Thus, five of the 10 patients operated upon in 1939 were more than 40 years

TABLE III

Blood Pressure Response in Respective Hypertensive Groups

Grade	Number of Patients	Effect of Operation on Blood Pressure	
		Slight and Considerable	No Response
1			
2	38	21 (55%)	17
3	8	3 (37%)	5
4	6		6

of age. Excluding the two postoperative deaths in Group B, only one patient has died; death took place two months after operation, of cardiac failure. Of the remaining 38 patients, 13 (34 per cent) had a satisfactory drop in blood pressure (table 2), 11 (26 per cent) had a slight drop in pressure, and 15 (39 per cent) had no change in blood pressure whatever.

In 22 cases a family history of hypertension was found, either one or both parents having an elevated blood pressure. Of these 22 patients, 10 obtained a drop in blood pressure and 12 had no drop. A family history of hypertension, therefore, is of no value in the selection or rejection of cases for operation.

The grade of hypertension played a significant rôle in the operative result, as shown in table 3. Early in our experience it was found that patients with Grade 4 hypertension did not improve following operation. The blood pressure remained at the preoperative level and death followed in the natural course of the disease. Of the patients with Grade 2 hypertension, 55 per cent of 38 obtained a drop in the blood pressure. Of those with Grade 3 hypertension, only three (37 per cent) of eight patients were benefited.

Most patients studied had Grade 2 hypertension, which accounts for the large number in this group having operation. No patient with Grade 1 hypertension was submitted to operation.

From our experience it seems certain that age is of considerable importance in selecting patients who are likely to obtain a satisfactory drop in blood pressure. Of 20 patients more than 40 years of age, only two had a drop in blood pressure. Of 32 patients less than 40, 22 (68 per cent) had a drop in blood pressure, there being 10 failures. The high percentage of

TABLE IV

Kidney Biopsies

Relationship of Kidney Disease to Drop in Blood Pressure

	Number	Number
Normal		4
Drop in blood pressure	4	
No drop in blood pressure	0	
Pathologic		21
Drop in blood pressure	11	
No drop in blood pressure	10	
Total		25

failures in 1939 when five of the 10 patients selected were more than 40 years of age is thus accounted for, whereas the larger number of successes in 1940 may be based on the fact that the patients were in the younger age group.

Symptomatic relief was found to occur in many cases even when a drop in the level of the blood pressure did not take place. This observation has been made by other investigators and is often an incentive to submit patients to operation even when a drop in the blood pressure is not likely to occur. Of 37 patients analyzed from the standpoint of symptoms, 27 had complaints related to hypertension, chiefly headache and vertigo, and of these, 20 were completely relieved of symptoms; only one half of these obtained a drop in the blood pressure. All patients having a drop in the blood pressure were relieved of hypertensive symptoms. As noted, only 27 of 37 patients had symptoms of their hypertension; almost 30 per cent of the patients were without hypertensive symptoms. These patients were studied because an elevated pressure had been found during a routine physical examination.

Of the 36 patients having only the splanchnic resection, 24 did not have a drop in the blood pressure; 12 patients obtained a drop. Of 18 patients having nephro-omentopexy in addition to splanchnic resection, 10 had a drop in blood pressure. It is not possible at this time to determine the significance of these figures as the series is not large enough. Theoretically, nephro-omentopexy is a possible means of increasing the circulation to the kidney. We have not as yet had the help of postmortem pathologic material to confirm this. Animal experimentation on this problem has given proof as to this means of increasing circulation.

In 25 patients, a specimen of the kidney was taken at the time of the operation when exploring the kidney and adrenal or when doing the nephro-omentopexy. Four patients had normal kidney tissue (table 4); all of them obtained a drop in blood pressure, three to a satisfactory level. Of the 21 patients showing various vascular and glomerular changes, there was an equal number of satisfactory and poor results. This latter finding, therefore, has no definite prognostic significance as regards a possible drop in pressure.

Comment

Our experience with these 54 patients confirms the experiences of others, namely that surgery of the sympathetic nervous system may have an ameliorating effect upon the blood pressure of patients with essential hypertension. The best results were obtained in patients with Grades 2 and 3 hypertension, whose ages were less than 40 and whose blood pressures responded to sedation. A better method of selecting patients for operation for hypertension needs to be devised.

Of the patients more carefully selected, 13 (34 per cent) of 38 had an excellent result and 11 had a slight drop in pressure. The blood pressure of the remaining patients was not altered. Symptomatic improvement was found to occur in 71 per cent of the patients; in one half of these the blood pressure remained elevated. This operation, we believe, is justified when patients fulfill the criteria which we have selected for the procedure, and when they understand that satisfactory lowering of the blood pressure is obtained in about one third of the cases.

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PHYSIOLOGICALLY DIRECTED THERAPY IN PNEUMONIA *

By ALVAN L. BARACH, M.D., F.A.C.P., *New York, N. Y.*

THE use of chemotherapy has made even more important an etiological classification in the diagnosis and treatment of pneumonia. Most cases of lobar pneumonia are due to organisms whose growth is stopped by sulfathiazole or sulfadiazine; in many instances similar organisms are the cause of bronchopneumonia. However, bronchopneumonia also occurs as a primary disease, the cause of which is obscure.[1, 2] The hypothesis considered most likely in a group of cases observed at the Presbyterian Hospital in the years 1938 and 1939 was that of virus etiology.[3] As Kneeland pointed out in 52 carefully analyzed cases, chemotherapy had no effect on the disease, whereas supportive measures, especially inhalational therapy, were of marked value. There are other types of bronchopneumonia which occur after operations and in the course of chronic illness, such as heart disease and asthma, in which inhalational therapy may be considered of special value, since respiratory or cardiac function is frequently impaired under these circumstances.

The purpose of this communication is to point out that recent advances in physiologically directed treatment may crucially alter the course of patients with bronchopneumonia or lobar pneumonia who do not respond to chemotherapy.

METHODS

The measures employed include (1) the administration of positive pressure, (2) inhalations of helium-oxygen mixtures, and (3) inhalation of the vaporized solutions of neosynephrine and epinephrine.

Oxygen is administered under positive pressure for the treatment of edema of the lungs, either as a result of increased permeability of the capillary endothelium, as in pneumonia or irritative gas poisoning, or as a complication of heart failure.[4] The application of positive pressure to the inner surface of the lung exerts an opposing pressure on the external capillary wall, tending to prevent the exudation of serum. In addition, there is some retardation of the entrance of blood into the lungs. Because of the latter circumstance, the one contraindication to the use of positive pressure is peripheral circulatory failure, namely shock, in which the venous return to the right heart is already impaired. Positive pressure is also used in the treatment of severe cases of asthma or in cases of bronchiolitis in which there is marked narrowing of the lumen of the smaller bronchi; helium-oxygen mixtures are employed in this type of obstructive dyspnea either with or with-

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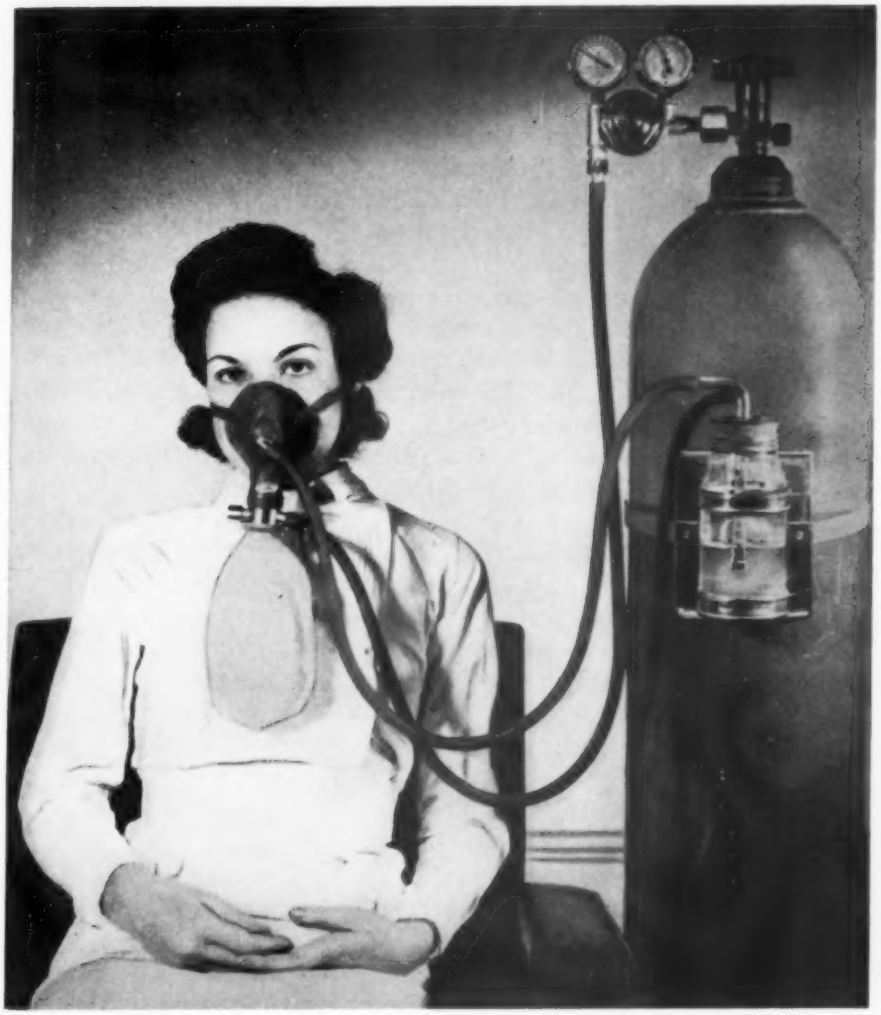

FIG. 1. Oxygen meter mask with attachment to water bottle to provide pressure during expiration.*

out positive pressure.[5] The most effective method of administering positive pressure is by the helium-oxygen hood in which the head of the patient is enclosed by a hood with a transparent plastocele window. Pressures of 1 to 6 cm. of water are maintained both in inspiration and in expiration.[6]

Positive pressure may be administered in expiration only by the use of a mask in which the exhaled air passes outward through a tube which is immersed to a variable degree under water. The injector mask,[7] which may be employed both for administration of oxygen in percentages of 40 to 100

* The injector mask apparatus is made by the Oxygen Equipment Mfg. Co., 405 East 62nd Street, New York, N. Y.

per cent and also for inhalation of helium-oxygen mixtures, may be adapted for the administration of 100 per cent oxygen under positive pressure of 1 to 5 cm. of water. In order to accomplish this the expiratory valve is removed and an adaptor containing the rubber tubing is put in its place, as shown in the accompanying illustration. Under ordinary circumstances it is generally not necessary to use pressures above 3 to 4 cm. of water. The pressure may be gradually lowered by lifting the glass connecting tube from 4 to 3 and then to 2 or 1 cm. of water as desired.

Although helium-oxygen mixtures are most effectively given by means of the hood, especially under positive pressure, the mask illustrated above may be used. It is better to admit a large flow of the helium-oxygen mixture, such as 7 to 10 liters per minute on the oxygen regulator, than to attempt to economize. In oxygen therapy the collecting bag should be kept about half expanded at the end of inspiration, but in the administration of helium-oxygen mixtures for the treatment of asthma there is even less resistance if the bag is kept almost full both during inspiration and expiration.

The inhalation of the vaporized solutions of neosynephrine and epinephrine is best obtained by passing 5 liters of oxygen from a high pressure tank through a nebulizer in which has been placed 1 c.c. of a 1 per cent solution of neosynephrine and 0.5 c.c. of a 1 : 100 epinephrine solution. Any of the nebulizers on the market may be used, the hand bulb being detached and the rubber tube from the oxygen tank being inserted in its place. Neosynephrine is used for its vasoconstriction effect on the mucous membrane of the tracheobronchial tree and 1 : 100 epinephrine as a bronchodilator.[8, 9]

The following case histories illustrate the type of case treated, the methods employed, and results of treatment.

Case Reports

Case 1. A woman of 58 years had suffered from numerous attacks of acute bronchitis in one of which bronchial asthma had occurred. Present illness began with a cold, followed by increasing cough, dyspnea and prostration. The temperature mounted to 103° F., pulse to 120, and the respiratory rate to 28. The lungs contained sibilant râles scattered throughout the chest, moist râles at both bases, more marked on the right. The sputum revealed no pneumococci on repeated smears and cultures, nor any hemolytic streptococcus organisms. Blood count revealed 11,000 white blood cells, polymorphonuclears 75 per cent. An intermittent fever continued for two days, 101° to 103° F., preceding chilly sensations. Sulfathiazole was administered for three days, a total of 19 grams being given. At the end of this time the patient was weak and prostrated, and difficulty in breathing became pronounced. On the third day inhalations of 1.0 c.c. of 1 per cent neosynephrine and 0.5 c.c. of 1 : 100 epinephrine were begun at three hour intervals. These substances were vaporized by passing 5 liters of oxygen through a nebulizer containing the mixed solutions. Following each inhalation there was subjective relief and objective evidence of decrease in the number of sibilant râles. On the fourth day inhalations of a mixture of 25 per cent oxygen and 75 per cent helium were given for 45 minutes after each spray. The mask used was the injector meter mask which contains an inspiratory valve to prevent re-

breathing and accumulation of carbon dioxide. More marked and prolonged relief of wheezing respiration followed each helium-oxygen inhalation.

A portable roentgen-ray of the chest at home on the fourth day of disease showed nothing except increased bronchial markings. Expectoration of 120 c.c. of mucopurulent secretion was examined daily and showed no significant organisms, merely *Streptococcus viridans* and *N. catarrhalis* organisms. On the fifth day of disease she was put in an oxygen tent, with a transparent pliofilm canopy covering the entire bed, and an oxygen concentration of 60 per cent was maintained. Two hours later her breathing was less labored, the respiratory rate declined from 28 to 24, and she fell asleep. Although the intermittent fever continued for four more days the patient was now comfortable, with little dyspnea, prostration or weakness. After five days the tent was removed and helium-oxygen inhalations discontinued. Two days later the temperature became normal. The neosynephrine-epinephrine sprays were decreased to twice daily during the following two weeks and then stopped. Several minutes after spraying the patient generally brought up large gobs of mucus which she was previ-

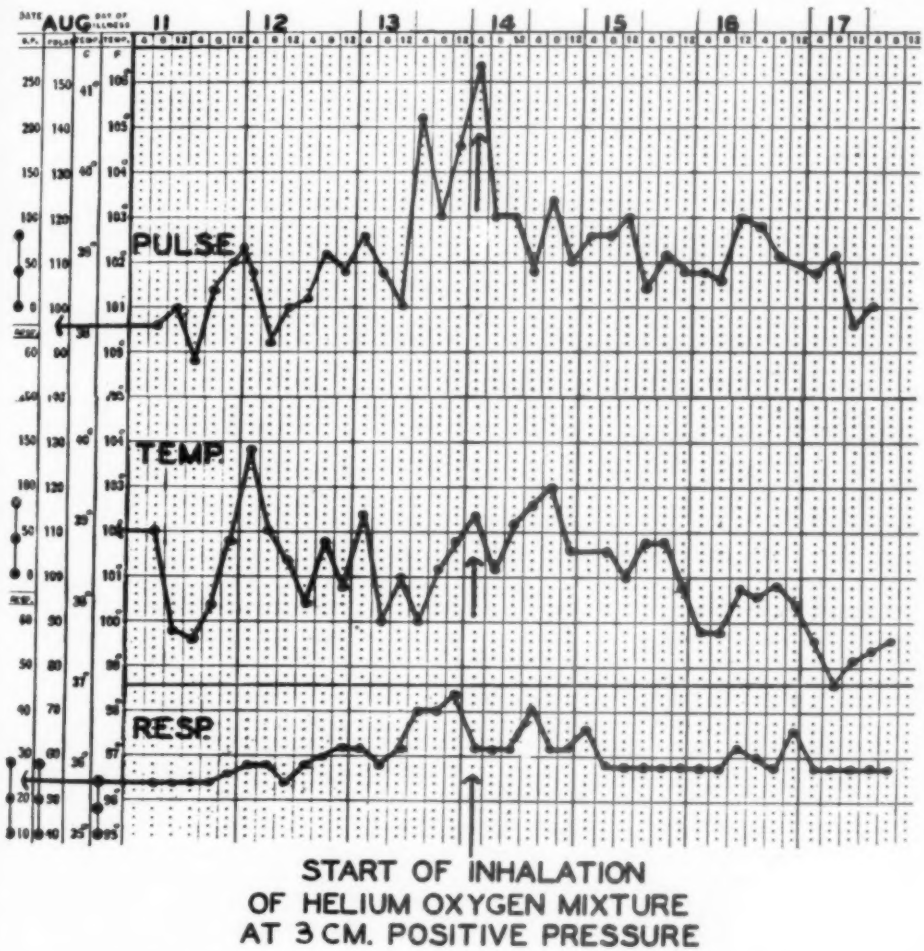

FIG. 2. Effect of helium oxygen inhalation under positive pressure in a case of bronchopneumonia with pulmonary edema.

ously unable to expectorate even after consciously provoked coughing. The spray appeared to facilitate expectoration of mucus which may have been adherent to the bronchial wall or caught by tight bronchial spasm.

Additional treatment consisted of ingestion of saturated solution of potassium iodide 1.0 c.c. four times daily, aminophylline 0.2 gm. four times daily, and codeine phosphate 0.03 gm. four times daily. On two occasions aminophylline was also given rectally for persistent bronchial spasm, 0.6 gm. in 20 c.c. of tap water, inserted by catheter and syringe, in each instance with further amelioration of bronchial spasm.

The diagnosis was bronchopneumonia of undetermined etiology, probably belonging to the group of so-called "virus" pneumonia. The inhalation of the neosynephrine-epinephrine sprays and helium with oxygen markedly reduced the severity of obstructive dyspnea and aided in expectoration of sticky mucus. Continuous residence in 60 per cent oxygen was of conspicuous and almost immediate benefit, decreasing the volume of breathing, lessening fatigue and prostration, and aiding sleep.

Case 2. Male, 30 years of age. The patient had had asthma as a child, but this had not returned in recent years. The present illness began with chilly sensations, malaise, and weakness of two days' duration, followed by a dry cough and a rise in temperature to 104° F. On examination he was found to be an acutely ill, well-developed male, in respiratory distress; the lips and nail-beds were slightly cyanotic. Over the right upper lobe a few coarse râles were heard. Blood count revealed 9,800 white blood cells, polymorphonuclears 73 per cent. The sputum showed *Streptococcus viridans* predominating on the first examination, and on the second test, a pneumococcus type 18 was isolated. He was given sulfapyridine without effect for three days. During this time he developed increasing cyanosis, dyspnea and cough, with signs of asthma which were unrelieved by hypodermic administration of adrenalin or intravenous injection of 50 per cent glucose. The roentgen-ray at this time showed consolidation in the right upper lobe and in the left upper lobe. On the seventh day after admission he was put in an oxygen tent, with a concentration of 50 per cent oxygen. Although his color was slightly improved, bronchial asthma persisted and gradually became increasingly severe. He was then placed in a helium-oxygen hood with 30 to 40 per cent oxygen, the remainder helium, under a positive pressure of 5 cm. of water.

This brought immediate and dramatic relief to the dyspnea from which he had suffered up to this time. When the pressure within the hood was lowered from 5 cm. to 3 cm., the patient immediately experienced an increasing difficulty and distress in breathing. The administration of positive pressure was obviously essential for the maintenance of comfort as well as respiratory function. For nine days he required helium-oxygen therapy under positive pressure. At the end of this time the pressure was gradually lowered and he was placed in an oxygen tent with 55 per cent oxygen. He made a slow, gradual improvement with intermittent fever over the next two weeks, and from then on gradually recovered strength.

The diagnosis was bronchopneumonia of undetermined origin, probably in the virus group. The importance of positive pressure in this case was amply demonstrated by both the subjective feeling of distress and also by the appearance of difficult breathing when it was withdrawn. Inhalational therapy enabled the patient to overcome a most severe type of obstructive dyspnea for a period of nine days. This case, as well as the succeeding one, were in the group reported by Kneeland.[3]

Case 3. Male, aged 29. The patient entered the hospital with a 24 hour history of cough and fever without headache or chill. On examination the lungs were clear. Temperature was 102° F. Blood count revealed 6,500 white blood cells, polymorphonuclears 69 per cent. Three days after admission he felt chilly, and his temperature rose to 104.4° F. Roentgen-ray of the chest showed a lobular pneumonia in the left lower lobe. Nine days after admission temperature was 104° F., pulse 110, respiratory rate 40. He was weak, toxic and cyanotic. He was placed in an

oxygen tent, oxygen concentration 50 per cent, and felt more comfortable until a new patch of pneumonia developed in the right lung field. Asthmatic wheezing and a constant impulse to cough added to his distress. He was now markedly dyspneic and cyanotic. The oxygen concentration was elevated to between 70 and 80 per cent, and oxygen sprays of the vaporized solutions of neosynephrine and epinephrine were given at two to three hour intervals, with marked relief. Removal from the tent atmosphere revealed the extent to which the high oxygen atmosphere was maintaining respiratory function, since severe dyspnea and cyanosis took place immediately each time he was taken out of the tent for the following four days. At that time he began to improve noticeably and oxygen therapy was stopped two days later. He made a slow gradual recovery.

The sputum culture at no time showed pneumococcus organisms. *Streptococcus viridans* predominated. Diagnosis was bronchopneumonia, probably of the interstitial or virus type, with severe diffuse involvement, profound oxygen-want, and bronchial spasm.

Case 4. A woman of 57 years became ill with cough, malaise, and intermittent fever to 102° F. On examination she was stout, moderately comfortable, with a respiratory rate of 24 and a pulse of 96. The lungs showed a few moist râles at the bases and scattered sibilant râles. During the next four days the respiratory rate gradually increased to 44, and breathing became more labored, with persistence of sibilant râles and finally the development of widespread bubbling moist râles in both lungs, heard also in the throat. Overt pulmonary edema was now present, with a pulse of 154. She was markedly dyspneic, cyanotic and in acute distress. The patient was first treated with inhalation of the vaporized spray of 1.5 c.c. of 1 per cent neosynephrine and 1 c.c. of 1:100 epinephrine, administering 5 liters of oxygen per minute through the nebulizer. The helium-oxygen hood was then applied with a concentration of 40 per cent oxygen and 60 per cent helium under a positive pressure of 3 cm. of water.[6] After the spray and administration of the helium-oxygen mixture under pressure the severe dyspnea was markedly relieved; the moist râles in the lungs began to clear within half an hour and were entirely absent after eight hours. As shown in the accompanying chart, the pulse rate fell the same day from 154 to 110, although the temperature still continued high at 103° F. Respiratory rate decreased from 44 to 32. For the next three days the patient was kept in the helium-oxygen hood and during this period sprays of the neosynephrine-epinephrine mixture were given at three to four hour intervals. At the end of three days she was comfortable, the helium-oxygen tent was removed, and she made an uneventful recovery.

This patient demonstrated that positive pressure is useful, not only for the maintenance of a patent airway, but also for the treatment of pulmonary edema and threatened asphyxia. The mechanism of the use of the positive pressure in the treatment of pulmonary edema has been previously described.[4] The diagnosis was bronchopneumonia of undetermined origin, bronchial asthma, and pulmonary edema.

Case 5. The patient, a 31-year-old female, developed cough, fever, and increasing shortness of breath following a cold of one week's duration. Examination within 24 hours of onset of these symptoms disclosed beginning signs of consolidation in the left lower lobe. Temperature was 103.8° F., pulse 120, and respiratory rate 36. Examination of sputum was negative for pneumococci by the Neufeld typing method, but two cultures out of four revealed type 23 and type 30. Sulfadiazine was administered by mouth and parenterally for five and a half days, the average daily dose amounting to 5 grams. Although the temperature gradually returned to normal after four days, the patient continued to be desperately ill. On the third day of illness outspoken pulmonary edema was present, with loud bubbling râles heard in the throat and moist râles heard on auscultation throughout both chests. For a period of 36 hours repeated intravenous injections of 50 c.c. of 50 per cent glucose held the edema in check temporarily. On the fifth day of the disease the patient was stuporous, and

breathing was labored with a deep inspiratory retraction of the lower sternum. Moist râles were heard over both chests, front and back, and there were bubbling râles in the trachea. For three days the patient had been in an oxygen tent with a concentration of 50 per cent oxygen.

The oxygen concentration in the tent was elevated to 70 per cent for three hours by administration of 30 liters of oxygen per minute without effect on her condition. She was put in a positive pressure hood with 100 per cent oxygen and a positive pressure of 3 cm. of water. Within one hour the bubbling throat râles had disappeared and no signs of moisture were heard in the left anterior chest region. There was also marked diminution of the râles in the right anterior chest. At 4 a.m. the following morning the stopper of the water bottle which regulated the pressure had slipped upward so that the patient was receiving 1 cm. of water pressure. Three and a half hours later the signs of edema had returned throughout both lungs. One hour after raising the pressure to 3.5 cm. both the bubbling throat râles and the moist râles over the anterior chest disappeared. The patient remained in the helium-oxygen hood for three days during which time the pressure was gradually lowered to 1 cm. During the last 24 hours she was given 70 per cent oxygen with 30 per cent helium in the hood for a total period of seven hours. She was then placed in an oxygen tent with a concentration of 70 per cent oxygen. During the next three days this was gradually lowered to 40 per cent when the tent was removed.

The day after the positive pressure treatment was begun it was found that she had consolidation of both lower lobes and the right upper lobe. On the third day after positive pressure therapy the patient ran a low fever which gradually returned to normal during the next five days.

The diagnosis was thought to be a pneumococcus lobar pneumonia, probably complicated by "virus" infection. The effect of inhalation of 100 per cent oxygen under a positive pressure of 3 to 3.5 cm. of water was followed by unmistakable and prompt disappearance of the signs of widespread pulmonary edema. Other treatment included the administration of sugar and salt by Levine tube inserted through the nose, the inhalation of the vaporized sprays of 1 per cent neosynephrine and 1 : 100 epinephrine at three hour intervals. Neosynephrine 0.5 c.c. was mixed with epinephrine 0.5 c.c. and vaporized by running 5 liters of oxygen through a nebulizer which was held in the patient's mouth.*

The value of positive pressure in the treatment of edema of the lungs following operations is shown in an interesting report by Boothby, Mayo and Lovelace.[10] On the sixth day after cholecystectomy, their patient developed widespread pulmonary edema with bubbling tracheal râles which could be heard on entering the room. No reduction in the signs of edema took place on administration of 100 per cent oxygen, but 20 minutes after application of a positive pressure of 6 cm. of water no râles were audible in either lung. The administration of 100 per cent oxygen was then continued, and the patient made an uneventful recovery.

Discussion

Narrowing of the bronchial lumen occurs not only in asthma but is frequent in pneumonias of undetermined origin, especially in the so-called "virus" group.[1, 2, 3] The obstruction is not only due to bronchial spasm but to edema of the bronchial wall and to accumulation of tenacious mucopurulent secretion in the small branches of the tracheobronchial tree. In-

* This case is reported through the courtesy of Dr. Russell Cecil.

halation of oxygen-enriched mixtures counteract to a variable extent impaired diffusion of oxygen and irregular ventilation of the alveoli, resulting in a decrease of the volume of breathing, lessened dyspnea and cyanosis. When the obstructive factor in dyspnea is prominent, inhalation of helium-oxygen mixtures under positive pressure decreases the mechanical effort of breathing and helps to maintain a patent airway. The application of positive pressure to the inner surface of the lung also counteracts the tendency to edema of the lungs, in lobar pneumonia as well as bronchopneumonia. Inhaling the sprays of the vaporized solutions of 1:100 epinephrine and 1 per cent neosynephrine reduces bronchial obstruction by stopping spasm of the bronchial wall, loosening tenacious mucus, and by vasoconstriction of the mucus membrane of the tracheobronchial tree.

Summary

Four cases of bronchopneumonia of undetermined origin and one case of lobar pneumonia are reported in which physiologically directed therapy was of crucial value in the treatment of anoxia, respiratory obstruction and pulmonary edema. The measures employed included (1) application of positive pressure, either in conjunction with 100 per cent oxygen or helium-oxygen mixtures, (2) inhalation of the vaporized solutions of epinephrine and neosynephrine, (3) helium-oxygen inhalations, and (4) continuous administration of high oxygen concentrations in tents with completely transparent canopies. The importance of maintaining respiratory function in pneumonia unresponsive to specific drug therapy was illustrated by the response of these cases to the various technics of inhalational therapy described.

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AN OXYGEN MASK METERED FOR POSITIVE PRESSURE *

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In the preceding paper [1] the clinical advantage of breathing oxygen or oxygen-helium mixtures under positive pressure was described. When the head of the patient is placed within a hood that is made leaktight at the neck by a soft sponge rubber collar, positive pressure may be applied to the inner surface of the lung during inspiration and expiration.[2] Since the technical management of a hood requires more careful supervision than is ordinarily available, we have added to a mask previously described [3] a mechanism which provides positive pressure during the expiratory cycle.

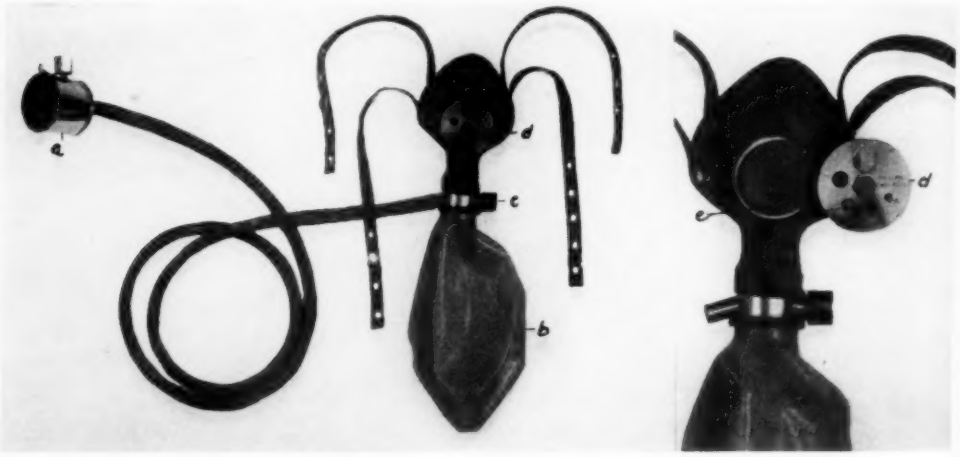

Fig 1. *a*. Air mixer.
b. Collecting bag.
c. Emergency intake valve.
d. Adjuster for control of expiratory pressure.
e. Expiratory flutter valve.

This consists of a series of apertures which are smaller than the diameter of the larynx. When expiration takes place through a constricted orifice, a positive pressure is reflected backward into the lung, the extent of the pressure being determined by the size of the orifice and the pulmonary ventilation. In the accompanying picture a dial is shown with five openings. When the largest opening is used there is little or no pressure during expiration. The other orifices have been calibrated during quiet breathing in an

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adult having a pulmonary ventilation of 5.5 liters per minute, so that pressures of 1, 2, 3, and 4 cm. H_2O are obtained when turned to the appropriate opening. Within this dial a flutter valve is situated which prevents the outside atmosphere from entering the mask, irrespective of the size of the opening on the outside of the dial. This mask is thus metered for positive pressure.

Barach [4] previously suggested that physiological advantages attended grunting and groaning in such instances as lobar pneumonia, bronchial asthma and pulmonary emphysema. Measurement in a tracheotomized patient of the degree of pressure developed during a forceful grunt revealed pressures as high as 30 cm. H_2O. Not only does it appear likely that the expiratory grunt maintains a more patent airway, as suggested by roentgenological studies,[5] but it also exerts an opposing force on the capillary wall which tends to stop or to reduce the passage of serum from the pulmonary capillaries into the alveoli.[6]

In this mask an injector is attached to the oxygen tank to provide a measured concentration of oxygen in the inspired air. A bag of light latex is used to collect oxygen, being separated from the mask by an inspiratory valve which prevents rebreathing. Another addition to the mask is an emergency inspiratory valve which opens when the bag is collapsed. Thus, even if a shortage of oxygen takes place for a limited period, no sensation of distress will occur since the inspiratory valve operates under minimal pressures.*

Summary

A mechanism which provides positive pressure in expiration is described. By turning a dial to varying sized orifices, expiratory positive pressures may be administered from 0 to 4 cm. of water. A flutter valve behind the disc makes for leaktight closure during expiration, irrespective of the size of the orifice.

Positive pressure in expiration may be used for the treatment of pulmonary edema due to increased hydrostatic pressure and anoxia, as in heart failure, or to changes in permeability of the pulmonary capillaries, as in pneumonia or gas poisoning. Positive pressure is not indicated in those conditions characterized by a deficient venous rate to the heart, such as shock.

The oxygen mask apparatus with or without positive pressure may be employed for the delivery of oxygen-enriched atmospheres from 40 to 100 per cent or for the administration of helium-oxygen mixtures. An emergency inspiratory valve has been added in order to prevent any sensation of distress if the collecting bag is temporarily collapsed.

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FUNCTIONAL MITRAL STENOSIS *

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A sign commonly considered characteristic of mitral stenosis is a mid-diastolic murmur at the cardiac apex. The murmur is usually low-pitched, rumbling, and not very loud. It is separated from the second heart sound by a definite time interval. It may be short in early diastole or may extend throughout diastole. In the latter case it decreases at first in intensity to increase again toward the end of diastole (presystolic murmur). It is usually well localized, near the apex, less commonly at the mitral valve area.

Murmurs indistinguishable from the diastolic murmur of mitral stenosis have been described in a variety of other conditions: Aortic regurgitation (Austin Flint murmur),[1] adhesive pericarditis,[2] various types of chronic anemia with heart enlargement,[3] and rheumatic heart disease in young people without the presence of organic mitral stenosis.[4, 5]

We have observed this murmur in young patients suffering from acute nephritis with hypertension and cardiac dilatation, disappearing when blood pressure and heart size returned to normal. Recent studies on the heart in nephritis fail to mention this murmur.[6] We shall report these cases and attempt to find a common basis for the explanation of the diastolic murmurs occurring under different conditions.

CASE REPORTS

Case 1. A negro boy, 13 years of age, was brought to the hospital because of convulsions and generalized edema. Four weeks prior to admission he had a sore throat, followed within 24 hours by a generalized erythema. The rash and fever disappeared after a few days and at the end of the week the skin began to desquamate. Three days before admission he had a generalized convulsion. At the same time the legs and face began to swell. He became drowsy and weak. During the night preceding admission he had 9 or 10 convulsions. Physical examination showed a well developed colored boy. He was difficult to arouse and showed Cheyne-Stokes respiration. The face and lower extremities showed some edema. The skin was still desquamating. The throat and tonsils were somewhat injected and there were many sordes around the mouth. Auscultation of the lungs revealed many coarse moist râles throughout. The heart appeared somewhat enlarged, the left border of dullness extending 9 cm. from the midline in the fifth interspace. The heart sounds were distant. A rather blowing diastolic murmur was heard near the apex but was not transmitted. The heart rate was 88, respirations 24, blood pressure 170 mm. Hg systolic and 130 mm. diastolic, temperature 100. The urine showed a two plus albumin, many erythrocytes, leukocytes and granular casts. The blood showed a mild secondary anemia. A diagnosis of postscarlatinal nephritis and hypertensive encephalopathy was made.

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Treatment consisted of oral and intramuscular administration of magnesium sulphate, restriction of salt and protein but not of water. The blood pressure fell to 120 mm. Hg systolic and 80 mm. diastolic within six days and the cerebral symptoms disappeared. The temperature dropped to normal, the heart size decreased, and the diastolic murmur disappeared within one week after admission, although the urinary changes persisted for several months.

Case 2. This patient had been suffering from severe temporal headaches, pain across the chest and gradually increasing dyspnea following a severe sore throat. He was seen in the outpatient department four weeks after the onset of his illness. His blood pressure was found to be 180 mm. Hg systolic and 120 mm. diastolic. He was given digitalis. During the following two weeks the dyspnea improved but some edema of the face developed. He had a "fainting spell" three days before admission to the hospital. Following this "spell" he had complete relief from headaches.

Physical examination on admission showed a well developed negro boy, 13 years of age, in no apparent distress. There was slight edema of the face. All lymph nodes were moderately enlarged. The heart was somewhat enlarged toward both sides, M. R. being 3 cm., and M. L. 10 cm. in a teleroentgenogram. There was a systolic murmur at the aortic and pulmonic areas. Both the second aortic and pulmonic sounds were markedly accentuated. There was a soft diastolic mitral murmur heard and recorded. Exercise increased the intensity of the murmur. The heart rate was 100, the blood pressure 140 mm. Hg systolic and 80 mm. diastolic, the respirations 20 per minute. The lungs were entirely normal. The urine was essentially normal. The blood showed a moderate anemia (red blood cells 3,900,000; hemoglobin 75 per cent). An electrocardiogram showed low T_2 and late inversion of T_3 suggesting myocardial damage. A cold-pressor test showed a rise of blood pressure from 138 mm. Hg systolic and 80 mm. diastolic to 160 mm. systolic and 70 mm. diastolic but was essentially negative when repeated a few days later. Treatment consisted of salt free diet and continuation of digitalis, 0.1 gm. daily. The patient recovered from all symptoms in the course of two weeks. The diastolic murmur disappeared. No general diagnosis was made.

Case 3. This patient was admitted to the hospital for generalized edema of three or four days' duration. Physical examination showed a well developed negro boy, six years of age, showing slight dyspnea and generalized edema. Tonsils and cervical lymph nodes were moderately enlarged. The heart was somewhat enlarged to the left, the apex extending 1 cm. beyond the midclavicular line. A diastolic murmur was heard over the mitral area and demonstrated by a stethogram. The heart rate was 70 per minute, the blood pressure 150 mm. Hg systolic and 95 mm. diastolic. There were signs suggestive of moderate ascites. The lungs were clear. The urine showed a moderate albuminuria, a few leukocytes per low power field, but no erythrocytes or casts. The blood showed 68 per cent hemoglobin and 3,800,000 erythrocytes. Roentgen-ray of the chest showed moderate general enlargement of the heart and slight mottling outside the cardiac area. The electrocardiogram showed a low T-wave in Lead I. Treatment consisted of bed rest and intramuscular injection of magnesium sulphate on admission. The blood pressure dropped to 118 mm. Hg systolic and 70 mm. diastolic on the second day; the edema disappeared within three days; the diastolic murmur was not heard after the fifth day. The urine became normal after 10 days. The child was dismissed on the twenty-third day, after another roentgen-ray showed normal heart and lungs.

Case 4. This patient, a seven year old negro boy, was brought to the hospital for edema of the face and lower extremities. Three weeks before admission he contracted a severe cold and sore throat, but recovered completely within a few days. Four days before admission he injured his left thumb and contracted a superficial skin infection. On the following day the scrotum began to swell and one day later the mother noticed

edema of the face which increased in intensity until admission. Physical examination showed a well developed boy with moderate edema of the face, anterior abdomen, genitalia, and thighs. The heart showed definite enlargement, the left border extending 2 cm. beyond the midclavicular line. There was a rough systolic murmur heard loudest over the mitral area and transmitted to the axilla. In addition, there was a low pitched short diastolic murmur, not transmitted, heard best midway between the mitral area and the apex (figure 1a). The heart rate was 108 per minute, the blood

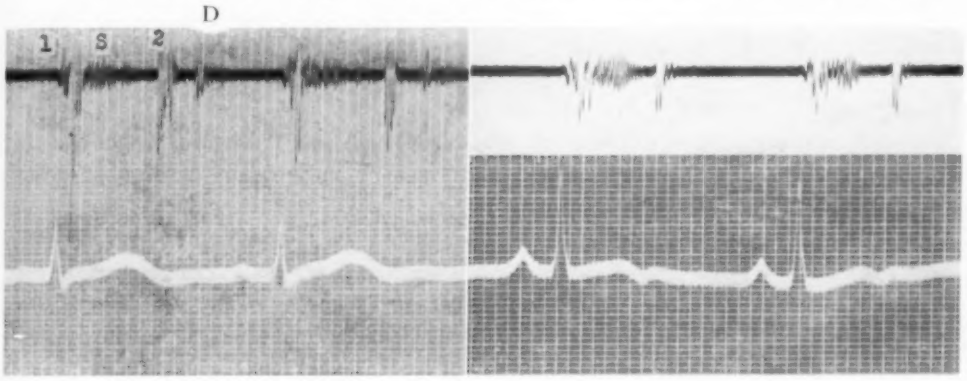

FIG. 1a. 1—first heart sound, *S*—systolic murmur, *2*—second heart sound, *D*—diastolic murmur.

FIG. 1b. Note absence of diastolic murmur.

pressure 160 mm. Hg systolic and 120 mm. diastolic. The urine was free of albumin on admission, but showed small amounts during the following three days. Examination of the blood showed: leukocytes 20,000, erythrocytes 3,310,000, hemoglobin 8.4 grams. An electrocardiogram, taken two days after admission, suggested slight myocardial damage because of low T_3, "W" shaped QRS_3 and late inversion of T_4.

A tentative diagnosis of acute glomerulonephritis was made. Within four days the skin infection healed under surgical treatment. The edema disappeared; the urine became free of albumin; the blood pressure dropped to 98 mm. Hg systolic and 48 mm. diastolic; and the diastolic murmur could neither be heard nor demonstrated in a stethogram (figure 1b.) The patient was dismissed free of symptoms on the sixth day.

DISCUSSION

These four patients have several points in common. All four of them were young male negroes who gave a history of past good health and, notably, of absence of rheumatic heart disease; all four suffered more or less from acute hypertension and consequent cardiac dilatation; all four showed moderate anemia; and in all four cases rheumatic heart disease was suspected at some time during hospitalization.

The condition does not appear to be uncommon. These cases were observed during a period in which approximately 10 other cases of acute nephritis were seen in the hospital. These other cases did not show the diastolic murmur, although some of them did show signs of cardiac failure and dilatation.

The origin of the diastolic murmur may be tentatively explained as follows:

The formation of eddies in the flow of liquids through tubes is favored by increased velocity of flow, rapid change in diameter of the tube (diverging boundaries) and decreased viscosity of the liquid. The eddies produce vibrations which may be heard as murmurs. It is not known at present whether the valve cusps, chordae tendineae, papillary muscles or extracardiac structures contribute most to the vibrations. Conditions in mitral stenosis are favorable for the production of murmurs. Auricular pressure is high and the velocity of blood flow, even if the volume flow is decreased, is therefore rapid; the mitral orifice is narrowed, whereas the auricle, and sometimes the ventricle also, is dilated; and, finally, anemia is frequently present reducing the viscosity of the blood. Flow from the auricle into the ventricle is not uniform as may be seen from ventricular volume curves.[7] Flow is rapid in early diastole immediately after the opening of the A-V valves. The intensity of the murmur diminishes when auricular pressure decreases during the course of diastole and may again increase preceding systole when auricular contraction increases auricular tension.

Our patients presumably had dilated auricles and ventricles; the viscosity of their blood was decreased because of anemia; and their auricular pressures were high because of beginning heart failure. The mitral orifice in acute hypertension and in the other above named conditions is probably of normal size. However, it is possible as suggested previously [4] that the change in diameter of the tube is more important than the actual size of the opening. Murmurs are absent in small infants although the valve orifice is smaller than in most adults suffering from mitral stenosis. Murmurs may also be absent in pulmonary stenosis when the pulmonary artery is hypoplastic but, on the other hand, murmurs may be heard over aneurysms in the absence of any obstruction.

Summary

A mid-diastolic apical murmur was heard and recorded in four cases of acute transient hypertension in negro boys.

The possibility of an erroneous diagnosis of mitral stenosis in this condition has been pointed out.

An attempt has been made to explain the occurrence of the murmurs in different conditions by analyzing the hydrodynamics of flow through the mitral orifice, the major factor being the relatively small mitral orifice between acutely dilated auricle and ventricle.

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THE ASSOCIATION OF ATROPHIC GASTRITIS WITH HYPOTHYROIDISM; A PRELIMINARY REPORT OF 11 CASES *

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IT is well recognized that anemia and hypochlorhydria or achlorhydria are frequently present in patients having hypothyroidism or myxedema.[1] Gastroscopy has demonstrated that atrophy of the gastric mucosa is also frequently associated with anemia and hypochlorhydria or achlorhydria.[2]

This frequent occurrence of anemia and hypochlorhydria or achlorhydria in both hypothyroidism and atrophic gastritis led me to believe that it would be worth while to study a series of hypothyroid or myxedematous patients to determine whether or not there is a significantly high percentage of gastric atrophy present in these conditions.

Herein are reported a series of 11 cases of hypothyroidism and myxedema examined at the Colorado General Hospital and associated Out-Patient Clinic, in which examination of the stomach has been made gastroscopically or microscopically.

To save time and space, only data which seem significant to this study will be presented, and non-related findings, both positive and negative, will be omitted as far as possible.

CASE REPORTS

Case 1. L. L., female, aged 28, entered the Medical Clinic September 8, 1939 with a multitude of complaints, among which were nervousness for six months, several menstrual periods each month for the past six months, numbness of the extremities irregularly for several months, anorexia, nausea and "gas" on the stomach, occasional vomiting, abdominal pains, and constipation all her life. There had been no weight loss. The onset of most of these symptoms occurred after the death of her mother, upon whom she was excessively dependent. An operation for chronic appendicitis was performed in 1934, and in 1935 for adhesions. The medical and gynecological examinations revealed no definite abnormalities. Pulse was 76, regular. Blood pressure was 106 mm. Hg systolic and 66 mm. diastolic. Provisional diagnoses of psychoneurosis and functional uterine bleeding were made.

Laboratory examination. Blood count: hemoglobin 12.5 gm., red blood cells 4,250,000 per cu. mm., white blood cells 6,000 per cu. mm., polymorphonuclears 54 per cent, lymphocytes 32 per cent, endothelial cells 2 per cent, and eosinophiles 12 per cent. Gastric analysis showed no free hydrochloric acid in either fasting specimen or after the feeding of alcohol. With histamine, the free hydrochloric acid was 16° and the total acidity from 13° to 60°. Basal metabolic rate was minus 29. The gastrointestinal roentgen-ray series indicated a constant deformity of the duodenal bulb suggesting ulceration. Gall-bladder visualization showed a slight sluggishness in function. Gastroscopic examination revealed a patchy atrophic gastritis of the body of the stomach.

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Case 2. A. B., female, aged 63, entered the Medical Clinic September 29, 1939, complaining of chills (apparently the sensation of coldness rather than a true chill), weakness and joint pains of three years' duration. In the system review, history of nausea and occasional vomiting was elicited. She had swelling of the ankles also. On physical examination, her skin was dry and pale, pulse 66, blood pressure 128 mm. Hg systolic and 80 mm. diastolic. Provisional diagnosis: Osteo-arthritis, hypothyroidism and atrophic gastritis.

Laboratory examination. Blood count: hemoglobin 12.0 gm., red blood cells 3,800,000 per cu. mm., white blood cells 6,600 per cu. mm., and normal differential count. Basal metabolic rate was minus 24. Gastric analysis showed achlorhydria on fasting and after alcohol, total acidity 19° to 42°. Gastrointestinal roentgen-ray series showed some deformity in the pyloro-duodenal region, probably due to an organic lesion, but no evidence of six hour residue. Gastroscopic examination made November 8, 1939 showed extensive atrophic gastritis of the body of the stomach, slight superficial gastritis, and one superficial erosion.

Case 3. M. B., female, aged 44, entered the Medical Clinic September 19, 1939, complaining of poor vision of the right eye, tremor (both of these due to a neurological abnormality which improved rapidly without any treatment other than thyroid), puffiness of the eyes, dyspnea and swelling of the ankles. She also noted that her speech had slowed considerably. She had a good appetite, no nausea or vomiting, was constipated, but had no abdominal pain. She was gaining in weight. She had irregular menstruation, one miscarriage, but no other pregnancies. The physical examination showed a three plus puffiness beneath the eyes. The skin of the face was pasty in appearance, dry and scaly over the body. There were supraclavicular pads of fat, palpable liver edge, and the ankle jerks showed delayed and prolonged decontraction phase. Provisional diagnosis (exclusive of neurological disease): Myxedema.

Laboratory examination: Basal metabolic rate was minus 48. Blood count: hemoglobin 14.0 gm., 4,270,000 red blood cells, 6,350 white blood cells, and normal differential count. Gastric analysis showed 24° to 36° free hydrochloric acid and 50° to 86° total acidity. The gastroscopic examination, November 17, 1940, showed small but definite areas of atrophic gastritis.

The patient's basal metabolic rate was brought up to plus 3 by December 11, 1939, under 2 grains of thyroid extract daily. She lived out of the city, and returned to her home at this time on this dosage of thyroid. On August 19, 1940, she returned to the clinic for a check examination. She was feeling well. Basal metabolic rate was minus 18, and the blood count showed 11 gm. hemoglobin, 4,400,000 red blood cells, and 7,500 white blood cells. Gastric analysis (fasting specimen only) showed 22° free hydrochloric acid and 43° total acidity. The gastroscopic examination showed slightly more extensive atrophic gastritis than at the original examination.

Case 4. E. P., female, aged 18, was first seen in the Medical Clinic August 4, 1937, having been referred by the obstetrical department. A severe anemia, hemoglobin of 9.3 gm., 2,940,000 red blood cells, 7,050 white blood cells, and a normal differential count were found. She responded moderately well to iron and liver (orally and intramuscularly). She returned September 17, 1937 after her delivery and was found to be moderately anemic, but she discontinued treatment after two months. She reëntered the clinic September 21, 1938 complaining of loss of weight, and in addition she was again found to be anemic. Treatment, consisting of iron, dilute hydrochloric acid and oral liver, resulted in only partial improvement of her anemia, so liver extract intramuscularly was begun. The patient again discontinued treatment. On November 1, 1939, she returned complaining of dyspnea and of being underweight. Complete examination was done and a slightly hyperchromic type of anemia found. Gastric analysis showed 0° to 48° free hydrochloric acid, and 12° to 59° total acidity. Basal metabolic rate was minus 19. Because of the persistent anemia, gastroscopic

examination was done November 27, 1939 and an extreme atrophic gastritis of the body of the stomach was found.

Case 5. G. R., female, aged 33, entered the Medical Clinic September 6, 1939, complaining of general run-down condition, fatigue, nausea, vomiting, choking sensation (attributed by the patient to a goiter), pain in the lower right quadrant, and cough. System review elicited the history of "canker" sores in the mouth, anorexia, flatulency, loss of 12 pounds in weight. There had been an appendectomy at 18. Physical examination showed no goiter; pulse 80, regular; blood pressure 140 mm. Hg systolic and 90 mm. diastolic; tenderness over the cecum and sigmoid colon. Provisional diagnosis of anxiety state, irritable colon and chronic bronchitis was made.

Laboratory examination. Blood count: hemoglobin 15.0 gm., red blood cells 3,750,000, white blood cells 6,500 and a normal differential count. Gastric analysis showed 12° free hydrochloric acid and 43° total acidity. Basal metabolic rate was minus 23. The gastroscopic examination made on December 4, 1939 showed patchy atrophic gastritis of the body of the stomach and slight superficial gastritis near the cardia.

Case 6. T. D., female, aged 45, entered the Medical Clinic September 12, 1939 with the history of having had pernicious anemia for nine years, complaining at this time of excessive bleeding following the extractions of teeth, and weakness. System review elicited swelling of the face and body at times, chilly sensation almost constantly, good appetite but soreness and bloating in the abdomen, constipation, frequent tarry stools. Physical examination showed a pale icteric appearance, and a dry and scaly skin. Pulse 65, regular, blood pressure 110 mm. Hg systolic and 70 mm. diastolic. There was a systolic murmur at the apex. Provisional diagnosis of pernicious anemia was made.

Laboratory examination. Blood count: hemoglobin 13.3 gm., red blood cells 3,250,000, white blood cells 4,600, and a normal differential. Prothrombin time was normal. Gastric analysis showed achlorhydria after histamine injection, with total acidity 16°, and three plus blood. Basal metabolic rate was minus 26. Blood cholesterol 230 mg. The gastroscopic examination on January 31, 1940 showed patchy atrophy of the stomach, many hemorrhages in the mucosa and three small polypi.

Case 7. E. L., female, aged 61, entered the Colorado General Hospital February 5, 1940. History obtained from the patient's daughter stated the patient had not been well for 25 years, gradually becoming weaker until she had been confined to bed and entirely helpless for the past month. She had had severe constipation, and for one month an abdominal ache which was relieved by the passage of flatus. She had been cold all her life, and had had scaly skin, dyspnea and edema for years. The physical examination showed a cold, dry scaly, pale skin, thick lips, eyes nearly swollen shut, pulse 60, regular, blood pressure 120 mm. Hg systolic and 80 mm. diastolic, distended abdomen, edema of the extremities. Reflexes showed delayed contraction and relaxation. The provisional diagnoses of myxedema, avitaminosis, and gastrointestinal disease were made.

Laboratory examination. Blood count: 9.5 gm. hemoglobin, 2,590,000 red blood cells, 2,400 white blood cells, and a normal differential count. Slight variation in size and shape of the red cells was noted. Basal metabolic rate was minus 41. The gastric analysis showed no free hydrochloric acid and not over 16° total acidity with histamine. The roentgen-ray showed enlargement of the heart. The electrocardiogram gave a typical myxedema picture. On March 4, 1940, the patient died. Postmortem examination was performed and the following diagnoses made: Severe hypothyroidism with myxedema; cardiac hypertrophy; bronchopneumonia; severe gastric atrophy, cause undetermined.

Microscopic examination of the stomach: "The mucosa is thin and acellular, revealing moderate capillary congestion. The gastric glands are decreased in number

and size, revealing distortion and shriveling. Slight exfoliation of the superficial epithelium is seen in some parts. The muscularis mucosa is thin but well defined."

Case 8. J. W., female, aged 53, an old patient in the clinic returned March 13, 1940 because of pain in the left lower chest and upper left abdomen anteriorly. On previous admissions the patient had had roentgen-ray evidence of gall-bladder disease and basal metabolic rate as low as minus 30. The physical examination showed no abnormality except moderate obesity. Pulse was 80, blood pressure was 132 mm. Hg systolic and 88 mm. diastolic.

Laboratory examination. Blood count: 15.0 gm. hemoglobin, 4,500,000 red blood cells, 7,700 white blood cells, and normal differential count. Gastric analysis showed 29° free hydrochloric acid, total acidity 36°. Basal metabolic rate was minus 24. Roentgen-ray of the gall-bladder showed delayed emptying. Gastroscopic examination July 27, 1940 revealed hemorrhagic atrophic gastritis of the cardiac end of the stomach. The second gastroscopic examination September 23, 1940, after two months of treatment, showed atrophic gastritis but no hemorrhages.

Case 9. I. L., female, aged 66, entered the Medical Clinic July 8, 1940, complaining of difficulty in walking straight, exhaustion and swelling of the eyes, since the fall of 1937. There were no definite abdominal symptoms. The patient had dyspnea and palpitation upon exertion. Physical examination showed a pale, dry skin, and marked supraclavicular pads of fat. Pulse 57, blood pressure 126 mm. Hg systolic and 76 mm. diastolic. Provisional diagnoses of cerebral arteriosclerosis, hypothyroidism and anemia were made. Blood count showed hemoglobin 12.5 gm., red blood cells 3,900,000, white blood cells 5,450 and normal differential count except for 8 per cent eosinophiles. Basal metabolic rate was minus 48, blood cholesterol 296 mg. Gastric analysis showed no free hydrochloric acid, 24° total acidity. Gastroscopic examination made July 30, 1940 showed atrophic gastritis with one hemorrhagic area visualized.

Case 10. B. M., female, aged 47, entered the Medical Clinic March 25, 1940 complaining of fatigue and weakness, pain in the back and shoulders, premenstrual pain, and irregular and excessive menstrual flow. Onset of symptoms was six years prior to admission, following a thyroidectomy (pre-operative diagnosis of hyperthyroidism). System review elicited the complaints of dryness of hair and skin, constant "chilliness," and slowing of speech and thinking. Physical examination showed a dry and scaly skin, swollen eyelids and lips, sparsity of hair and eyebrows, slowing of speech. Pulse 60, blood pressure 155 mm. Hg systolic and 105 mm. diastolic. Provisional diagnosis of postoperative myxedema was made. Basal metabolic rate was minus 36. Blood cholesterol was 272 mg. Blood count: hemoglobin 13.0 gm., 4,700,000 red blood cells, 6,600 white blood cells, and a normal differential. The patient was started on thyroid treatment, but discontinued coming to the clinic after two months. On August 7, 1940 she returned to the clinic, at which time the blood count showed 9.8 gm. hemoglobin, 3,150,000 red blood cells, 6,500 white blood cells. Gastric analysis showed the presence of free hydrochloric acid; quantitative analysis was not done. The gastroscopic examination was done September 13, 1940 and erosive atrophic gastritis of the body of the stomach was found.

Case 11. H. C., female, aged 49, entered the Medical Clinic July 17, 1940 complaining of pain and stiffness of the joints of her fingers and toes for the past year, nervousness the past few years, belching and occasional vomiting the past year, and constipation. Physical examination showed no abnormality except tenderness and swelling of the small joints of the hands and feet. Pulse was 64, regular, blood pressure 154 mm. Hg systolic and 100 mm. diastolic. Blood count showed 13.3 gm. hemoglobin, 3,900,000 red cells, 6,600 white blood cells, large number of lymphocytes (60 per cent). The gastric analysis showed 0° to 3° free hydrochloric acid and 5° to 20° total acidity. Basal metabolic rate was minus 23. Blood uric acid was 4.6 mg. per 100

c.c. The gastroscopic examination was done September 17, 1940 and diagnosis of mild superficial and probable atrophic gastritis was made.

Before discussing these cases, it will be of value to tabulate the findings as to the severity of the hypothyroidism (as evidenced by the basal metabolic rate and the presence of myxedema), the blood findings, gastric acidity and the presence or absence of digestive or gastrointestinal complaints.

Case	B.M.R.	Myxedema	Anemia	Gastric Acidity	Gastro-intestinal Symptoms
1	Minus 29		+	Low	+
2	Minus 24		+	Achlorhydria	+
3	Minus 48	+	0	Normal	0
4	Minus 19		+	Normal	0
5	Minus 23		+	Low	+
6	Minus 26		+	Achlorhydria	+
7	Minus 41	+	+	Low	+
8	Minus 24		0	Low	+
9	Minus 48	+	+	Achlorhydria	0
10	Minus 36	+	+	Present (quantity not determined)	0
11	Minus 23		+	Low	+

Arranged as to myxedematous cases and simple hypothyroidism cases:

Myxedema	Anemia	Acidity	Gastrointestinal Complaints
1	0	Normal	0
2	+	Low	+
3	+	Achlorhydria	0
4	+	Present (quantity not determined)	0
Hypothyroidism	**Anemia**	**Acidity**	**Gastrointestinal Complaints**
1	+	Low	+
2	+	Absent	+
3	+	Normal	0
4	+	Low	+
5	+	Absent	+
6	0	Normal	+
7	+	Low	+

Eleven cases are too small a series from which to draw too definite conclusions in regard to the incidence of gastric atrophy in hypothyroidism, but the high correlation found in this group of patients is significant enough to justify publication of this preliminary report in the hope that it will stimulate other observers to look for this correlation in the cases which come under their observation. The number of cases of hypothyroidism or myxedema in which examination of the gastric mucosa can be made will not be large in the average clinic, and it probably will take considerable time before sufficient cases have been observed to determine accurately how high is the correlation between hypothyroidism and gastric atrophy.

The diagnosis of atrophy of the gastric mucosa made gastroscopically has many pitfalls since it depends usually upon a brief inspection of the gastric mucosa by a single observer, but in this work I have attempted to prevent this tendency to subjective error by having my observations checked in as many cases as possible by one or two other observers, and in cases 3 and 8 the patients were examined on two different occasions. The most convincing evidence, however, is the data obtained from case 7, in which the atrophy of the gastric mucosa was proved microscopically.

The possibility that the gastric atrophy might be due to an independent coincidental disease of the gastrointestinal tract rather than hypothyroidism was considered, especially as two-thirds of these cases had gastrointestinal symptoms. However, of the more definite cases, the four true myxedemas, three had no gastrointestinal symptoms and the fourth had only slight gastrointestinal complaints, so I believe this possibility is untenable.

With regard to the high incidence of digestive complaints in these cases, the report of Bassler [3] is of great interest. "Masked hypothyroidism may be the underlying cause of serious gastrointestinal disturbances and may imitate symptoms of chronic involvement of abdominal structures." "The gastrointestinal symptoms as arrayed by Ramsey are: anorexia, flatulence (especially after meals), constipation, occasional nausea and rare vomiting, gastric hypoacidity and achlorhydria, and abdominal pains which may simulate gastro-duodenal ulcer, cholecystitis and appendicitis." The symptoms described above are consistent with those of atrophic gastritis,[2] and the supposition that such atrophy does exist in this type of case would do much to explain the underlying mechanism of such gastrointestinal symptoms as observed by Bassler.

These cases have also been examined with the fact in mind that anemia does at times alter the basal metabolic rate.[4] This effect varies, at times raising the rate, at times lowering it. However, the average basal metabolic rate of those cases in this series having anemia is minus 30, which is more than would be expected from anemia alone; and of the total series, two of the cases did not show any anemia.

Speculation as to why gastric atrophy should occur with hypothyroidism suggests various possibilities. The most simple explanation would seem to be that the slowing up of metabolic processes occurring in hypothyroidism reflects itself by decreased activity and replacement of cells of the gastric glands,[5] and that the gastric atrophy is a direct result of the hypothyroidism. The anemia which is found so frequently in hypothyroidism could, by this hypothesis, be explained in two ways: first, on the basis of decreased activity of the blood forming tissues, as the direct result of inadequate stimulation by the thyroid gland; secondly, the anemia might be secondary to the gastric atrophy, which in itself predisposes to anemia, rather than due directly to thyroid deficiency.

Mansfield [6] has produced evidence for the existence of a myelotropic hormone derived from the thyroid and found in fresh thyroid tissue but

not present in desiccated thyroid or thyroxine. In view of this work, it seems possible that it is the lack of a specific myelotropic hormone of the thyroid in some or all cases of hypothyroidism rather than a non-specific depression of metabolism which accounts for the anemia present so frequently in these cases.

With the ever increasing number of hormonal substances being discovered, I believe it is not unlikely that the atrophy of the gastric mucosa may also be due to a deficiency of a specific "gastrotropic hormone" rather than to a non-specific effect of thyroid deficiency.

Case 3 seems to favor the theory that the anemia and the atrophy of the stomach are not due entirely to the thyroxine deficiency, for when this case was observed for the second time after eight months of fairly adequate thyroid therapy, it showed even more extensive atrophy of the stomach than at the first examination and for the first time showed a lowered hemoglobin.

If such a gastrotropic hormone should be found to exist, it should open up a promising new approach to the search for the still unknown basic cause of anemias associated with gastric atrophy (both macrocytic and hypochromic types), and combined cord degeneration. Perhaps the relationship of atrophy of the stomach to hypothyroidism may furnish a clue to the discovery of such a hormone.

Summary

Gastroscopic (and in one case, microscopic) examinations have been made of the gastric mucosa in a series of 11 cases of hypothyroidism and myxedema.

Atrophy of the gastric mucosa was diagnosed positively in 10 cases; probable atrophy was diagnosed in the eleventh case. No definite conclusions can be drawn from such a small series, but the hypothesis that hypothyroidism and myxedema are accompanied by a high incidence of gastric atrophy seems worth further investigation.

The possibilities have been suggested that the gastric atrophy and anemia may be a direct result of hypothyroidism, or that there may be, in cases of hypothyroidism, an associated deficiency of myelotropic hormone [6] and a deficiency of a postulated "gastrotropic hormone" to account for the anemia and gastric atrophy.

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PARENTERAL USE OF THE SULFONAMIDES

A CLINICAL AND EXPERIMENTAL STUDY*

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The efficacy of the sulfonamide drugs in the treatment of many infections is generally accepted. The Council on Pharmacy and Chemistry has listed certain sulfonamides, stating their action, use, toxicity, and general status. It must be emphasized, however, that these chemicals are still relatively new and, therefore, require further experimental study in some phases of their behavior. At present in the United States, oral or intravenous administration is favored, although in England [1, 2] and Canada [3] the intramuscular route is also used. We have studied the rapidity of absorption and secretion of the sulfonamide drugs, the degree of acetylation, and toxic effects, both general and local. Our observations indicate that different patterns of effect are produced by different routes of administration. This article presents a comparative study of different routes of administration and discusses the therapeutic advantages and safety of the intramuscular route.

Procedure

The following observations were made on six groups of average hospital patients, as well as on three normal healthy volunteers who took all six tests. Some of the subjects had mild infections at the time; others were convalescent; most of them were ambulatory. None had fever, and none of the subjects had received sulfonamide therapy for at least a week previous to the experiment. All subjects were on a regular house diet, and fluids were allowed as desired.

In the first group each subject received a single dose of two grams of sulfapyridine by mouth, in the second two grams of sodium sulfapyridine intravenously, in the third two grams of sodium sulfapyridine intramuscularly. Those of the fourth group received two grams of sulfathiazole orally, the fifth group two grams of sodium sulfathiazole intravenously, and the sixth group two grams of sodium sulfathiazole intramuscularly. The intravenous doses were given as a 5 per cent solution of the drug in distilled water, and the intramuscular doses were given as a 33 1/3 per cent solution. The intramuscular dose was given deeply into the gluteal muscles as a single injection of 6 c.c. of the solution.

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From the University of Nebraska, College of Medicine, Department of Medicine.

Blood levels were determined at frequent intervals throughout the following 24 hours, and the total urinary excretion of the drug for 24 hours was observed. Free and conjugated drug in the blood and urine were determined by the Marshall-Litchfield method, using the Cenco Photelometer with a Wratten No. 74 green filter.

Separate groups of subjects were used for each procedure, but since the curves of the three volunteers who went through the entire series fell into the same general pattern as did the others, we feel that the heterogeneity of the groups does not appreciably affect our results.

Results

Tables 1 and 2 summarize the data concerning blood levels, giving the average figure for each group as well as the range of individual readings. Curves of the average blood concentrations for each group are shown in figure 1.

There was considerable variation among individuals, even upon intravenous administration. In one subject the maximum blood concentration following an oral dose of sulfapyridine was not reached until the eighth hour, whereas the average peak for the group occurred at four hours. The narrowest range and, therefore, the most consistent results among individual subjects occurred with intravenous sodium sulfapyridine.

Maximum concentration in the blood was reached in two to four hours after oral and intramuscular medication. Intravenous injection brought an immediate high concentration, which fell rapidly to a level below oral and

Table I

Blood Concentration in Milligrams Per Cent Following the Administration of Two Grams of Sulfapyridine

	Time	Average of Group		Individual Range	
		Free	Total	Free	Total
Oral	2 hr.	1.7	1.9	0.6–3.1	0.7–3.1
	4	2.3	3.0	1.5–3.4	1.9–3.7
7 Subjects	6	1.5	2.5	1.0–1.9	2.1–3.3
	8	1.4	2.4	0.8–2.3	1.9–2.9
	10	1.4	2.2	0.6–2.0	1.7–2.8
	24	0.6	0.9	0.2–1.1	0.5–1.3
Intramuscular	1 hr.	1.4	1.5	0.4–1.6	0.6–2.5
	3	2.2	2.8	1.5–3.4	2.0–3.5
	5	2.1	3.0	1.2–3.6	1.8–4.0
11 Subjects	7	1.8	2.5	0.9–3.3	1.7–3.5
	24	0.4	0.8	0.2–0.8	0.4–1.1
Intravenous	5 min.	5.4	5.5	3.4–8.7	3.5–8.7
	1 hr.	3.0	3.5	2.0–4.0	2.4–4.4
	2	2.4	3.2	1.9–3.2	2.4–4.2
5 Subjects	4	1.7	2.5	1.6–1.8	1.7–3.7
	8	1.1	2.0	0.8–1.3	1.5–2.4
	24	0.4	0.9	0.1–1.0	0.2–1.6

TABLE II

Blood Concentration in Milligrams Per Cent Following the Administration of Two Grams of Sulfathiazole

	Time	Average of Group		Individual Range	
		Free	Total	Free	Total
Oral	1 hr.	2.2	2.3	1.2–2.7	1.3–2.7
	2	2.6	2.7	2.4–3.0	2.5–3.1
	4	2.6	3.1	1.4–4.0	1.8–4.6
4 Subjects	6	2.2	2.7	1.0–3.6	1.5–4.3
	8	1.6	1.9	0.8–2.6	0.9–3.1
	24	0.3	0.6	0.2–0.4	0.2–1.8
Intramuscular	1 hr.	1.3	1.5	0.3–2.0	0.5–2.2
	3	2.5	2.8	1.8–3.1	2.4–3.3
	5	2.5	2.9	1.7–3.3	2.7–3.3
7 Subjects	7	2.3	2.7	1.7–2.5	2.0–3.1
	9	1.9	2.1	1.3–2.3	1.7–2.4
	24	0.5	0.6	0.3–0.7	0.4–0.8
Intravenous	1 hr.	5.1	5.4	3.0–9.0	3.0–9.0
	2	3.7	3.8	2.3–6.2	2.4–6.4
7 Subjects	3	3.4	3.7	2.0–5.5	2.3–6.0
	5	2.3	2.6	1.5–4.0	1.6–4.4
	24	0.2	0.3	0.2–0.4	0.2–0.4

intramuscular readings within three or four hours. The intramuscular route produced approximately the same maximum level as that attained by oral administration; absorption was slightly slower, however, and an effective blood level was maintained for a longer period of time. Sulfathiazole, by all three methods, gave a higher peak than the same dose of sulfapyridine, and the rate of absorption was more rapid. Maximum blood concentrations of sulfathiazole were maintained somewhat longer than those of sulfapyri-

TABLE III

Percentage Excretion in the Urine for First 24 Hours Following the Administration of Two Grams of the Drug

	Sulfapyridine			Sulfathiazole	
	Average	Range		Average	Range
Oral	48	30–75	Oral	78	68–90
Intramuscular	50	38–75	Intramuscular	68	55–85
Intravenous	40	25–85	Intravenous	87	79–96

dine, producing a flat peak followed by a plateau, in contrast with the sharp peak of the sulfapyridine curve. This was true of both oral and intramuscular methods of administration. The blood concentrations at the end of 24 hours were approximately the same for all three methods.

The degree of acetylation was considerably greater after sulfapyridine than after sulfathiazole. With both drugs the percentage of the conjugated form in the blood after a single dose rose to a maximum at six to seven hours

and then fell steadily. The degree of acetylation was roughly the same by all three methods.

Table 3 shows the percentage of drug recovered from the urine in 24 hours. There was great variation among individuals, although the relative amount of sulfathiazole recovered was consistently much greater than of sulfapyridine.

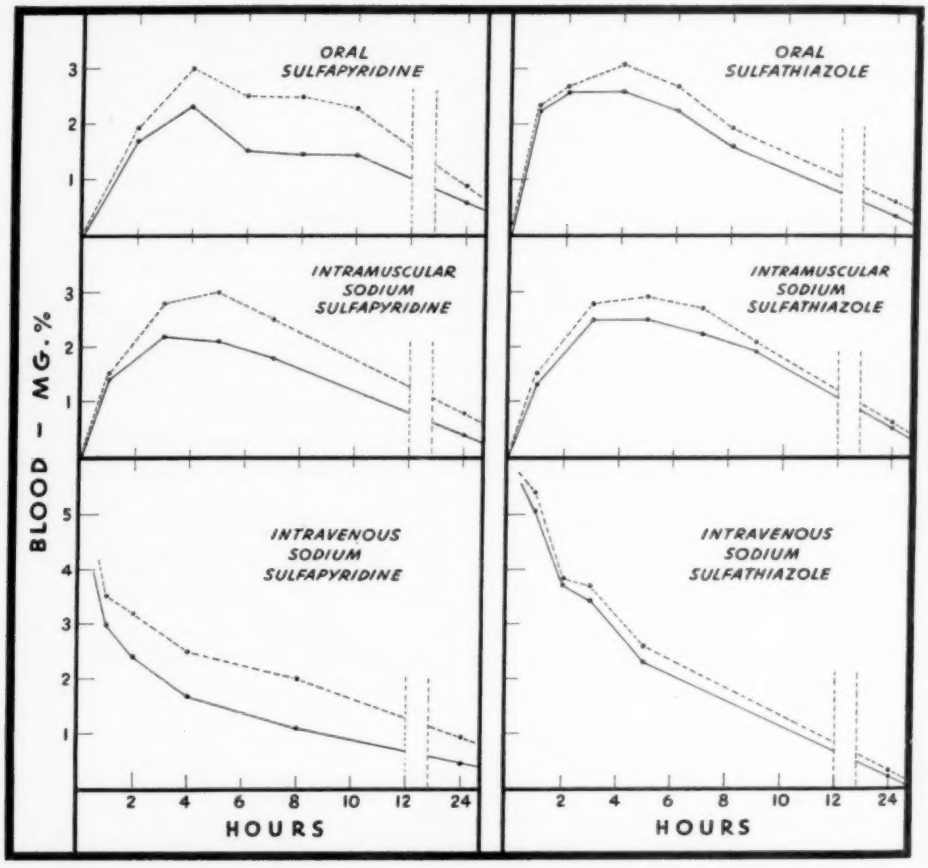

FIG. 1. Curves of free and total blood concentrations following the administration of two grams of sulfapyridine or sulfathiazole by various routes.

There were no toxic reactions except for occasional mild nausea and one instance of vomiting an hour after administration of an intravenous dose of sodium sulfapyridine.

CLINICAL OBSERVATION

We have previously reported the clinical use of intramuscular sodium sulfapyridine in a series of 25 cases.[4] Since that time we have enlarged this series to over 200 cases, representing more than 1500 intramuscular injections and including also the use of intramuscular sodium sulfathiazole in the same manner. Recently we have used sodium sulfadiazine intramuscu-

larly in a small series of cases with equally good results. Many of the patients received their medication by the intramuscular route exclusively; in others the intramuscular method was substituted for oral administration for a varying number of doses. This group of patients represented various types of infections for which sulfonamide medication would ordinarily be indicated; pneumococcal infections constituted the largest portion of the group.

The drug was prepared freshly each day as a 33 1/3 per cent solution in distilled water and injected deeply into the gluteal muscles or into the muscles of the thigh beneath the fascia lata. Originally our maintenance dosage schedule was 3 c.c. of the solution every four hours, but more recently we have maintained effective blood levels on a régime of 6 c.c. at six or eight hour intervals. In some cases isolated intramuscular injections were substituted gram for gram for oral medication.

Clinical results paralleled those obtained with oral medication. Toxic effects were no more frequent after intramuscular than after oral administration. In the group in which the intramuscular route alone was used, the incidence of dermatitis, hematuria, and leukopenia was somewhat lower than has been our experience with the oral route.[5] The most striking effect was the marked decrease in the incidence of nausea and vomiting (7.4 per cent). In several instances of severe nausea and vomiting after oral administration, relief was obtained when the change was made to intramuscular medication.

In the entire series two necrotic sloughs followed intramuscular injection. In both the drug was given, unsupervised, by a nurse with a short needle into the subcutaneous fat. In no case of correct administration into the muscular tissue was there any necrosis or inflammation. The degree of discomfort with the procedure was no greater than is seen with many drugs commonly given by the intramuscular method. Neither in rabbits nor in human subjects have we been able to demonstrate signs of inflammation or degeneration on microscopic examination of muscular tissue taken after intramuscular injection of sodium sulfapyridine.[5]

Discussion

In general, our oral and intravenous studies agree with results of other investigators,[6, 7, 8] both in general outline of the curves and great variability among individuals. Our purpose, however, has been to include the action of the drugs after the intramuscular method of administration and to compare the efficiency of this route with others.

Approximately the same blood concentrations are obtained by the intramuscular and oral routes, a fact shown in our clinical use of the method. Effective blood levels are maintained longer by intramuscular injection than by oral administration, thereby permitting a wider spacing of doses. Clinically we obtained effective results on a six and even eight hour dosage schedule.

The variation among individual subjects prevented drawing conclusions about comparative urinary excretion by the different routes.

The striking decrease in the incidence of nausea and vomiting with intramuscular use reopens the question of local or central origin of this distressing complication. If nausea and vomiting after sulfonamide therapy were of central origin, one would expect the incidence to be the same for all methods of administration, provided equal blood levels were reached. However, the lowered incidence after parenteral use with equally high blood concentrations points very strongly to the conclusion that the principal etiological factor is local irritation of the gastric mucosa. Strickler and associates [9] have shown the drug to be present in the stomach in fairly high concentrations following intravenous administration of sodium sulfapyridine. This could account for the few instances of nausea and vomiting which are encountered with parenteral use of the drugs.

Frequently in the course of clinical practice it is found that oral administration of the sulfonamides is not practicable. In comatose or delirious patients, after certain types of abdominal surgery, and in many patients, especially children, with severe nausea and vomiting, oral medication is undesirable and often impossible. Under such conditions a practical and effective means of parenteral administration of the drugs is necessary.

There are several objections to intravenous use of the drugs. In spite of the high initial concentration the blood level falls rapidly and a therapeutic level is not maintained as long as after an equal oral or intramuscular dose. Should any of the solution be introduced into the surrounding areolar tissue, necrosis, abscess formation, and ulceration may occur. When sodium sulfapyridine was first introduced, Marshall and Long [10] pointed out the dangers of sloughing after intravenous administration. The technical difficulties of this route are obvious with small veins, as in infants, children, obese women, and elderly patients. These difficulties add to the danger of the perivascular supporting tissues being infiltrated with the drug. Moreover, the highly alkaline sodium salts of these drugs tend to sclerose the veins, and repeated injections often render them unfit for further use.

Certain precautions must be taken, however, in the intramuscular use of the sulfonamides. Because of their high alkalinity, the drugs may produce necrotic sloughs, if they are deposited into the subcutaneous tissue. Care must be taken to use a long needle and to deposit the entire dose into the muscle. Unsupervised injection cannot be given by one unfamiliar with the necessity for correct technic. British observers [11] report paralysis and foot-drop from damage to the sciatic nerve by direct irritation of the drug injected intramuscularly. A little care in choosing the site of injection to avoid the region of the sciatic nerve will prevent this complication. The intramuscular injection of any irritating substance should be made deeply into the body of the gluteus medius muscle at a point midway between the greater trochanter and the iliac crest. Thigh injection should be made on the lateral aspect of the mid-thigh region through the fascia lata into the vastus lateralis muscle.

We wish to emphasize that the intramuscular method of administering sulfonamides is seldom the route of choice. Oral administration of any drug is always to be preferred. However, when there are gastrointestinal complications, or when it is difficult, impossible, or impractical to give drugs intravenously, an alternative route is desirable. It has been our purpose to show that sulfonamides may be given into the muscles, thus providing an additional safe and effective route of administration.

Summary and Conclusions

1. Studies are presented comparing the absorption and excretion of sulfapyridine and sulfathiazole by intramuscular, oral, and intravenous administration.

2. Absorption of the drugs after intramuscular injection is adequate and compares favorably with other routes.

3. No conclusion can be drawn as to the comparative urinary excretion by the different methods of administration.

4. Clinical experience with the intramuscular route is reviewed.

5. Certain necessary precautions with the intramuscular use of these drugs are emphasized.

6. The intramuscular administration of the soluble sulfonamide drugs presents certain advantages over other methods and offers a safe and effective alternate route when used correctly.

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PAINFUL FEET *

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THIS article is written for the practicing physician who has had but little experience in orthopedic surgery but has patients who complain about their feet. If referring them to an orthopedic surgeon is out of the question it is up to him to offer relief if that is possible; and it is because so many of these cases can be relieved by the family doctor that suggestions are here offered as to how to proceed. A somewhat "sketchy" review of the anatomy, mechanics and functions of the foot will be followed by directions as to the examination, diagnosis and treatment.

For the purpose of this article it is necessary to keep in mind only the three principal axes of movement of the foot. They are: the transverse, through the ankle joint; the longitudinal, through the subastragaloid joint; and the vertical, through the mediotarsal joint. The astragalus articulates with the tibia and fibula above, the os calcis below and the scaphoid in front. The articular surface which fits into the mortise formed by the tibia and fibula is narrow in its posterior part so that, although there is no lateral movement with the foot at a right angle to the leg or in dorsal flexion, there is slight lateral movement with the foot plantar flexed. That is why a high-heeled shoe makes the ankle less stable and more easily subject to strain than does the low-heeled shoe. Except for very slight rotation in full plantar flexion, which need not be considered in this article, movement at the ankle is in an anteroposterior plane; that is, around a transverse axis passing from the tip of the external malleolus to a point one-half inch below the tip of the internal malleolus. The joint between the astragalus and the calcaneum is divided by the dense interosseous ligament. Movement at this joint is around an axis passing obliquely from in front and medially, backward and laterally. (In the left foot, about 1:30 to 7:30 on the watch dial.) The movements, therefore, are inversion and eversion and take place in the subastragaloid and astragaloscaphoid joints. The astragaloscaphoid articulation should be considered with the calcaneocuboid articulation as motion in these two joints is usually concomitant. These four bones make up the mediotarsal joint. Motion takes place about a vertical axis, abducting and adducting the forefoot on the hindfoot. The articulations of the cuneiforms and the metatarsals need not be here taken up.

The functions of the foot are support and propulsion. With the muscles relaxed the ligaments prevent displacement. They become tight when the normal limit of movement in any joint is reached. The muscles arrange the bones, i.e., place them in the proper relationship to one another for any purpose. In an active foot, in walking for example, the purpose or function of

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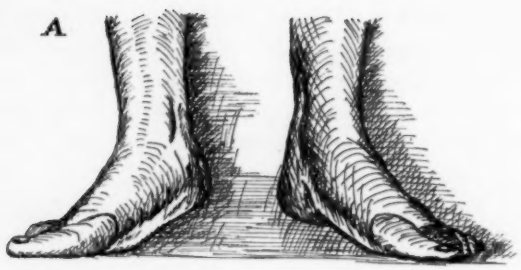
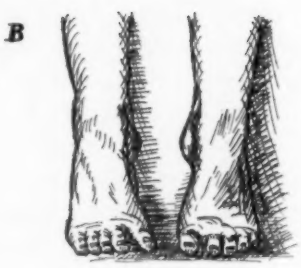
the foot changes a number of times, and the muscles must, therefore, change the bony relationship a number of times. In each step taken the foot receives the shock of contact with the ground, supports the body, raises the body and thrusts it forward, and is itself again brought forward, completing the cycle. Therefore, some groups of muscles direct the forefoot and hold the bones in the best possible relationship to receive the thrust of the body; others raise the hindfoot and others propel the body forward. Most of the muscles take part in each function to some extent. A fact to be noted here is that all physiological structures need both exercise and rest to be healthful—that is normal. Neither prolonged use nor prolonged rest is good for bone, ligament or muscle. With the foot at rest, as when one sits on a table with the foot hanging relaxed, it will be noted that the toe is slightly lower than the heel and the forefoot slightly adducted. In running, where this relaxed position is assumed after the foot leaves the ground and comes forward for the next step, the muscles do not become tired as quickly as in heel and toe walking. A dog-trot, for any distance, is less tiresome than walking. One is easily persuaded that heel-and-toe walking does jar the central nervous system. A man with a throbbing headache walks on his toes to avoid this jar and, in the absence of the headache, the jar must be there just the same. Hence, the adoption of rubber heels. The tap-tap-tap of high heels must be like slight but continuous hammer blows. This heel-toe walking is always used with pronated feet and it is hard to overcome; old habits are hard to change and most footwear prevents any change. With the foot in a stiff shoe the normal movements in the directions of eversion and inversion, abduction and adduction are impossible; to perform its normal functions the foot demands freedom of movement.

In standing, the ideal position for support of the body is with the feet parallel and about four inches apart. It is better to toe in markedly than to toe out ever so slightly. With the foot in abduction, the anteroposterior plane containing the center of gravity is brought to the medial side and thus away from the strongest part of the forefoot, tending at the same time to roll the foot downward on the medial side (eversion) and to push the forefoot into further abduction. This also puts a strain on the calcaneoscaphoid ligament and causes intra-articular pressure at the calcaneocuboid joint. As a result, pain may be felt about the strained ligament and compressed joint and callosities will form on the lateral side over the head of the fifth metatarsal and under the big toe joint from continuous pressure.

In walking correctly, the heel is raised and rotated inward and upward on the subastragaloid axis—the longitudinal axis—and thrust outward, by abduction at the mediotarsal joint, through the vertical axis. As the forefoot is held firmly to the ground the abduction is expressed in this movement of the hindfoot. Then, as the transverse plane of the body advances over the foot, the ground is spurned by the forefoot. When the foot is to be brought forward for the next step all of its muscles should be relaxed and,





A. Tip-toe position with toes pointing outward. All movements are confined to the ankle and the metatarsophalangeal joints. The foot is raised "en bloc."

B. Tip-toe position with feet pointing straight ahead. Every joint in the foot is functioning. The foot is plantar flexed, adducted and inverted. The forefoot being held fast to the ground, the movements are expressed mostly by the position of the hindfoot.

C. Wedges applied. Heel wedge raises up anteromedial corner of heel one-eighth, one-sixth or one-quarter inch. (Rarely one-half inch is needed.) Sole wedge does not raise medial side of sole. Its purpose is to fill in the space between the shank of the shoe and the floor, after the heel wedge is applied.

D. An inflexible shank made flexible by the removal of a wedge-shaped piece. The shank should be narrow as well as flexible. The extension of the wedges is also shown.

E. Strapping the weak foot with adhesive plaster. The foot is relaxed, the knee being

as explained above, the toe will be slightly in advance of the heel as it reaches the ground. In all athletic games the foot is used in this way, but modern shoes compel us to walk without using the functions of the foot except for dorsal and plantar flexion of the ankle joint and extension and flexion of the metatarsophalangeal joints.

An arthritis of one or more joints of the foot, as in any other part of the body, will cause a varying degree of immobilization through muscular spasm. Before instituting treatment a differential diagnosis must be made between a traumatic and an inflammatory arthritis. This is particularly true when the forefoot is immobilized in the position of abduction. Overcoming the deformity by stretching the spasmodic peronei, which is the correct treatment of a spasmodic flat-foot, would be erroneous for an infected astragaloscaphoid joint.

In the diagnosis and treatment of painful conditions of the feet a history of the case is of great value. If the feet were painless until recently, perhaps within a few weeks or even a few months, did a change in the amount the feet were used seem to cause the pain? If so, probably the feet were weak before but, because they were used but little, not much attention was given to the discomfort. A change in the style of foot apparel may be a cause, especially in the height of the heel. Are the feet painful upon first arising in the morning or are they comfortable until after walking a few blocks, and does the pain radiate up the backs of the legs? During the night the muscles are relaxed—they are not active in guarding the astragaloscaphoid joint from strain—and the pain is felt immediately when this joint becomes weight-bearing. After hobbling about a bit, the pain will lessen as the spasm returns and the joint is restricted in motion. Such a foot will be again painful after much use, perhaps in the afternoon of an active day. On the other hand, there may be no pain at all upon arising in the morning and none will appear until work for the day has been started. The patient will report having walked perhaps an eighth of a mile when his calves began to hurt so severely that he had to stop and even sit down. A frequent cause of this pain is lack of normal dorsal flexion of the foot, though there may be little or no deformity in any of the joints. Yet it must be borne in mind that these pains are sometimes the precursory symptoms of circulatory disturbances. Limitation of dorsal flexion, as will be shown later on, is the basic factor in many cases of weak and painful feet, and this condition should be suspected if the history shows that the patient feels that he must wear heels even on slippers, that high heels on shoes are more comfortable than

flexed to shorten the gastrocnemius, then held in dorsal flexion at a right angle, adducted and inverted. Three straps are used, each one and one-half inches wide and long enough to reach almost to the knee. The first strap is placed around the forefoot, its distal edge slanting to follow the base of the toes. It grasps the forefoot firmly and crosses the plantar surface from the lateral to the medial side and up the medial side of the leg. The second strap reinforces the first and overlaps it three-quarters inch. The third strap begins above the lateral malleolus, comes down, under the heel, and up the medial side of the leg. The purpose of the strapping is to check abnormal movements, thus teaching normal joint and muscular sense and supporting atrophied muscles.

low, that pain was referred up the leg and that backache was frequently present. Inquiry should be made as to how much the feet have been used, in athletics, long walks, or much standing.

Examination. When standing, with the shoes on, does the patient toe out, are the legs straight and the patellae facing forward (that is are the legs rolled outward from the hips) and, in rising on tip-toe, does he adduct the foot or is movement restricted to the metatarsophalangeal and ankle joints? Examining the shoes, note if the soles are worn more toward the medial side than laterally and where the heels are worn down at the back. If the wear has been on the lateral side it shows that the patient walks with the toes out. Are there longitudinal creases in the leather just back of the position of the great toe? Such creases are made by abduction of the forefoot with consequent lowering of the arch. Standing, in bare feet, are the sides of the heels vertical or are the outer sides sloping downward and outward? Are the feet abducted? Do the toes lies flat against the ground? Rising on tip-toe: are the mediotarsal joints adducted and are the plantar surfaces of the heel inverted? Sitting on a stool at a right angle to the patient, so that the patient's ankle may rest on the knee of the examiner, note any puffiness or swelling; the color of the skin, especially of the toes; the patency of the dorsalis pedis and the posterior tibial arteries; the location of all callosities. Callosities are due to pressure. If found under the heads of the five metatarsals, they are always due to a shortened heel cord; on the outer side of the foot, opposite the head of the fifth metatarsal, they are due to pressure of the foot against the shoe from abduction; beneath and to the medial side of the big toe they are due to pressure caused by eversion. The passive movements in the three principal axes are now tested. With the foot in slight inversion and adduction and the knee in full extension, dorsal flexion should be to 90 degrees or less. Abduction, adduction, eversion and inversion should be free, normal in range and painless. Is the calcaneocuboid or the astragaloscaphoid joint painful to pressure? Manipulate the toes to determine their freedom of motion. Freedom of movement of the first metatarsophalangeal joint is essential to comfort and its absence may be the chief factor in causing abduction.

Now if it is quite evident that, in standing and walking, the foot is used in the position of eversion and abduction, diagnosis of "weak foot" or "pronated foot," both being practically the same, may be made and treatment instituted. However, if there is evidence of circulatory trouble or heat or swelling or muscular spasm limiting any of the movements, then an exact diagnosis of the underlying cause of such a condition must be made before any other procedure. If the forefoot is held in abduction by spasm of the peroneals, hot foot-baths and absolute rest for a few days should restore free passive movement, and if they do not, roentgenograms should be taken and a careful differential diagnosis made between spasmodic flat-foot and bone or joint disease.

Treatment. With all movements free and normal in range, with the foot in the standing position in eversion, as shown especially by the heel, and in abduction, as shown by the forefoot being turned outward, treatment is directed toward restoring the foot to its normal position and keeping it there, at the same time forcing it to function normally. This is accomplished by altering the shoes, strapping the feet and obtaining the coöperation of the patient. The weakened muscles, especially the tibials, must be built up, the joint sense must be reëducated and normal function restored. A wedge is placed on the anteromedial part of the heel, its purpose being to restore the normal inclination of the calcaneum. It should be one-eighth to one-quarter inch at its thickest part, depending upon the degree of rotation of the calcaneoastragaloid joint. Usually, it is better to use a one-quarter inch wedge at first and reduce it as improvement progresses. A sole wedge is applied, filling in the space between the first metatarsal and the supporting surface. This may be flared medially, and the more it extends medially the more will it help to prevent eversion and abduction. A good cobbler can give a flare of at least one-sixth inch. This wedge does not tilt up the inner border of the foot as does a wedge along the medial part of the sole of a shoe. It allows the sole to lie flat on the ground in the same plane as the bottom of the heel *after the heel wedge is applied.* The sole wedge must be of such a length as to prevent the metatarsal from being lowered at its proximal end. From two to two and one-half inches is usually satisfactory. The shanks must be flexible, so flexible as to offer no resistance to the adducting and inverting of the foot. In these cases the muscles used in the movements of adduction and inversion are usually so weak that at first any resistance may be enough to prevent their action. Most shoes have a strip of steel in the shanks. This is removed and, if its removal does not make the shoe fully flexible, a V-shaped piece is cut out of the shank. The shank cannot be too flexible.

The reason shoe manufacturers do not usually make the shanks flexible is that most people still toe out, using only the ankle joint and the metatarsophalangeal joints, and consequently the weight at the midtarsal would soon distort and make ugly a shoe that was built with no stiffening in the shank. It is apparent, therefore, that this line of treatment is not appropriate for the patient who will not carry it out. Normal functioning of the foot demands freedom of use of all joints and, conversely, a foot used abnormally needs artificial support. Adhesive strapping is used to help to direct the weakened muscles and to reëducate the joint senses. For the first few weeks it will be necessary to keep the strapping constantly applied and thereafter it will need to be applied intermittently, to prevent the resumption of old habits. With the shoes wedged and the strapping applied, the patient is instructed always to use the foot with the toes pointing inward. The foot functions properly with the toes pointing straight ahead, but it is better to instruct the patient to try to toe in.

Specific exercises are unnecessary if the patient indulges in any sports which keep him on his toes. Dancing is splendid for weak feet. Standing is hard on the structures of the feet as they are subjected to continuous work and strain. It can be explained to the patient that every physiological structure needs both work and rest—the heart being a good example—and that, in standing, exhaustion can be prevented by constantly changing the supporting muscles and ligaments, by raising the heels slightly, then resting on both heels and soles, then standing on the outer borders of the feet. This, if repeated every few minutes, will be of great benefit.

There is one factor which is present in many cases of painful feet, which may be a cause of metatarsalgia and certainly is an obstacle to overcoming abduction, and that is a shortened heel cord. If dorsal flexion is arrested at 90 degrees, the arrested movement thrusts the weight of the body against the forefoot, like a blow at each step. If dorsal flexion is limited to 93 degrees or more, it is impossible to walk without abducting the foot, unless the heels are of such a height as to make dorsal flexion necessary to only 95 degrees.

If the above treatment is persisted in, the gastrocnemius will, in many cases, be stretched by use in the corrected position; if not, other methods must be used to lengthen the calf muscles.

It should be kept in mind by all physicians that pronated feet may be a contributing factor in faulty posture, backache and muscular pains of the lower extremities.

THE COMPARATIVE VALUE OF CALCIUM GLUCONATE, MAGNESIUM SULFATE, AND ALPHA LOBELINE HYDROCHLORIDE AS AGENTS FOR MEASUREMENT OF THE ARM TO TONGUE CIRCULATION TIME IN 50 PATIENTS WITH AND 50 PATIENTS WITHOUT HEART FAILURE*

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THE arm to tongue circulation time is now recognized as a valuable means of determining the efficiency of cardiac function. It is also useful in the study of other diseases in which the velocity of blood flow is altered. Specifically, it measures the time required for the passage of a drug from the point of injection in one of the veins of the antecubital fossa through the systemic veins, the right side of the heart, the pulmonary system, the left side of the heart, and the systemic arteries to a point of perception, usually the tongue.

In conditions in which the velocity of blood flow is diminished, the arm to tongue circulation time is prolonged. This occurs most commonly in congestive heart failure, so that in general the test has its greatest use in this condition. Its clinical value here lies in the aid afforded in confirming the diagnosis of heart failure in questionable cases and in following the progress of any case. On the other hand, the test is perhaps equally valuable in a negative way for excluding the diagnosis of heart failure in patients with dyspnea or other symptoms which suggest cardiac decompensation.[1] It should be noted that in certain cases of heart failure the circulation time may not be prolonged. Hyperthyroid heart disease [2] and beriberi heart disease [3] are the best known etiologic varieties which illustrate this.

Other causes for prolongation of the arm to tongue circulation time include conditions in which the venous return to the heart is obstructed, such as cardiac compression and local lesions of the superior vena cava or its tributaries. Polycythemia rubra vera [4] and myxedema [5] also cause generalized slowing of the velocity of blood flow.

The arm to tongue circulation time is decreased in conditions in which the blood flows more rapidly. This is true in hyperthyroidism,[2] severe anemia,[4] and fever. In hyperthyroidism, the test has obtained some popularity as a simple means of confirming the diagnosis.

The drugs used for measurement of the arm to tongue circulation time include calcium salts and magnesium sulfate which produce a sensation of warmth in the patient's throat and tongue, saccharine which causes a sweet taste, decholin which gives a bitter taste, sodium cyanide which causes a

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gasp when the drug reaches the carotid sinus, and lobeline which also acts on the carotid sinus to produce a paroxysm of coughing. Various reports [6, 7, 8, 2, 9, 10] indicate that all of these drugs give approximately the same results for measurement of the circulation time in normal persons. The range most usually quoted for the normal arm to tongue circulation time is from 9 to 16 seconds. Obviously in a strict sense, all of these agents cannot be said to measure the arm to tongue circulation time because not all of them have the tongue as their point of perception. However, for practical purposes it has been assumed that the terms, arm to tongue, arm to throat, and arm to carotid sinus, may be used interchangeably. In our opinion any one of these terms is preferable to the term, total circulation time, which lacks the anatomical definitiveness of the former. In this paper, the term, arm to tongue circulation time, has been adopted.

A drug that is to be used in clinical practice for measurement of the arm to tongue circulation time should fulfill certain requirements.[11] It should be non-toxic in the dose employed and should be known to have no undesirable effect on the pathological condition being studied. It should have a minimum of unpleasant side effects and should be eliminated rapidly enough so that it can be used repeatedly. It should have an end point which is easily recognized, and the number of cases in which the drug fails should be minimal. The drug should be readily available for general use and preferably should be inexpensive. All of the drugs mentioned above in connection with measurement of the circulation time fulfill these requirements adequately enough to justify their employment. However, saccharine and sodium cyanide are not readily available for general use except in large clinics or hospitals where they can be prepared inexpensively to order.

We have previously reported [12] observations which demonstrate that measurements of the arm to lung circulation time by means of ether may be significantly different from those obtained with paraldehyde in patients with heart failure. Kvale and Allen [11] have compared sodium cyanide and a calcium-magnesium-sodium solution for measurement of the arm to tongue circulation time. They found that in normal persons an inherent error of about three seconds exists in measurements obtained with sodium cyanide. It has been our interest, therefore, to compare the results obtained in measurement of the arm to tongue circulation time by means of three readily available agents, namely, calcium gluconate, magnesium sulfate, and lobeline. The object of this study has been to determine whether or not these three drugs would give equal results when used in the same patient. The demonstration has been attempted in 50 patients without apparent cause for alteration of the velocity of blood flow and in 50 patients with heart failure.

Technic. Measurement of the arm to tongue circulation time was obtained in each case after the patient had been resting in bed for at least one-half hour. The patient was in supine position at the time of the test, with the arm in which the injection was to be made abducted through an angle of 45 degrees and in such position that the veins of the antecubital fossa were at

or below the level of the right auricle. One of the veins was entered with a 20-gauge needle, 30 seconds were allowed to elapse after removal of the tourniquet in order to permit restoration of blood flow, and then 5 c.c. of 20 per cent calcium gluconate * were injected *as rapidly as possible*. The time from the beginning of the injection until the patient called "Now" at the moment of feeling a sense of warmth in the throat and tongue was measured by means of a stopwatch. A period of several minutes was then allowed to pass, in order for the sensation produced by the calcium gluconate to disappear. During this short period, the needle remained in the vein and a small amount of physiologic saline was injected slowly in order to prevent clotting within the lumen of the needle. Following disappearance of the feeling of warmth produced by calcium, 5 c.c. of 10 per cent magnesium sulfate were injected *as rapidly as possible* and the same procedure followed as for calcium. Again, after passage of a sufficient interval for disappearance of the sensation of warmth produced by the magnesium solution, 5 mg. of alpha lobeline hydrochloride * were injected through the same needle in the same manner. The time that elapsed from the beginning of the injection until the patient began a short paroxysm of coughing was measured with a stopwatch. In those instances in which 5 mg. of alpha lobeline failed to provoke coughing after an interval of about two minutes, 7.5 mg. of the drug were injected for a repetition of the attempt to measure the circulation time. In all cases, a careful watch was kept for other possible manifestations of reaction to the alpha lobeline, such as grimacing, hyperpnea, and gasping. The end point which was observed first was used for the circulation time measurement. In those cases in which the circulation time measurement by means of alpha lobeline seemed unusually shorter than the measurements obtained with the other drugs, it was suspected that the arm to lung circulation time had been measured. An attempt to confirm this suspicion was made in such cases by measuring the arm to lung circulation time with ether.

Clinical Results

The results of measurement of the circulation time with calcium gluconate, magnesium sulfate, and alpha lobeline in 50 patients who had no cause for a reduction of the velocity of blood flow are shown in table 1. The results of similar measurements in 50 patients with heart failure are shown in table 2.

In the 50 patients who did not have heart failure, there was no instance in which all three of the drugs failed to produce an end-point. There were 11 cases in which 5 mg. of alpha lobeline failed to give an end point. In all but three of these, 7.5 mg. of the drug were effective. However, there were two additional cases in which alpha lobeline yielded a record of the arm to lung circulation time, as shown by comparison with measurements made by

* Calcium gluconate as ampules of 20 per cent Neocalglucon and alpha lobeline hydrochloride in ampules of 1 per cent solution were supplied by the Sandoz Company.

TABLE I

Cases without Heart Failure

Case No.	Diagnosis	Arm to Tongue Circulation Time (Seconds)			
		Neocal-glucon	Magnesium Sulfate	Alpha Lobeline	
				5 mg.	7.5 mg.
1	Metrorrhagia	13.8	14.8	No end point	15.0
2	Prostatism	15.0	17.0	No end point	13.0
3	Hypertension	10.2	10.0	No end point	No end point
4	Hypertension	13.0	14.2	11.2	
5	Hypertension	12.0	15.0	10.6	
6	Nephritis	11.0	11.6	No end point	No end point
7	Carotid sinus syndrome	12.8	11.8	9.4	
8	Normal control	10.0	10.0	8.2	
9	Hypertension	No end point	14.0	(G)15.0	(G)15.0
10	Hypertension	14.6	14.6	No end point	No end point
11	Alcoholism	12.0	11.0	10.6	
12	Pulmonary tuberculosis	14.0	18.0	17.4	
13	Hypertension	10.8	11.0	10.8	
14	Prostatism	14.8	14.2	(G)15.0	
15	Hypertension	15.0	15.0	(G)14.0	
16	Hypertension	No end point	20.8	No end point	14.8
17	Rheumatic heart disease	15.6	16.0	15.0	
18	Parkinsonism	15.2	15.6	(G)13.0	
19	Rheumatic heart disease	15.0	13.6	12.2	
20	Cirrhosis	14.0	13.0	12.8	
21	Cerebral thrombosis	13.8	15.0	*6.0	
22	Hypertension	17.2	18.2	(G)10.0	
23	Carcinoma of pancreas	14.6	14.8	14.2	
24	Hypertension	15.0	15.4	12.0	
25	Hypertension	17.0	17.0	15.0	
26	Gastric carcinoma	15.0	15.8	11.0	
27	Hypertension	18.6	18.0	No end point	14.8
28	Hysteria	14.0	16.0	12.4	
29	Ménière's syndrome	10.2	12.2	*6.8	
30	Obesity	15.0	16.5	No end point	(G)13.0
31	Chronic glomerulonephritis	13.2	13.2	No end point	(G)13.0
32	Hypertension	18.0	18.0	14.0	
33	Hypertension	15.6	16.6	15.0	
34	Hypertension	16.0	16.8	11.2	
35	Bronchopneumonia	15.0	15.0	14.0	
36	Hypertension	17.0	16.2	16.0	
37	Hypertension	14.0	17.0	No end point	15.0
38	Diabetes mellitus	15.6	16.2	16.0	
39	Aortic aneurysm	16.0	14.0	No end point	(H)17.0
40	Pneumonia	11.6	11.4	9.2	
41	Pneumonia	12.0	12.4	10.0	
42	Alcoholism	14.0	14.2	12.0	
43	Hypertension	13.0	14.4	11.2	
44	Alcoholism	11.0	11.2	10.0	
45	Alcoholism	10.0	10.0	9.0	
46	Cerebral arteriosclerosis	11.0	11.6	10.0	
47	Manic-psychosis	10.0	10.2	10.0	
48	Alcoholism	12.8	13.0	11.0	
49	General paresis	12.0	12.4	11.4	
50	Alcoholism	14.0	14.0	12.2	

TABLE II

Cases With Heart Failure

Case No.	Etiologic Type of Heart Disease	Arm to Tongue Circulation Time (Seconds)			
		Neocal-glucon	Magnesium Sulfate	Alpha Lobeline	
				5 mg.	7.5 mg.
1	Rheumatic	37.5	41.0	32.5	
2	Hypertensive	19.2	19.0	23.0	
3	Hypertensive	19.2	20.4	18.0	
4	Hypertensive	No end point	71.0	No end point	No end point
5	Hypertensive	60.8	58.0	No end point	No end point
6	Hyperthyroid	26.0	23.0	18.0	
7	Hypertensive	No end point	19.0	No end point	18.0
8	Hypertensive myxedema	41.0	42.0	No end point	40.5
9	Hypertensive	18.0	18.0	18.0	
10	Hypertensive	45.0	41.0	No end point	*26.0
11	Hypertensive	23.8	24.8	23.2	
12	Coronary artery sclerosis	25.2	No end point	33.4	
13	Hypertensive arterio-sclerotic	28.0	24.0	No end point	21.0
14	Hypertensive	35.0	47.0	No end point	40.8
15	Hypertensive	49.2	61.8	No end point	(H)75.0
16	Hypertensive	27.2	25.0	*15.8	
17	Syphilitic	20.6	25.6	20.6	
18	Syphilitic	70.0	69.6	No end point	75.4
19	Rheumatic	19.0	17.0	No end point	15.6
20	Syphilitic	24.0	25.0	19.2	
21	Hypertensive	No end point	No end point	No end point	49.0
22	Coronary artery sclerosis	21.6	20.0	No end point	(G)23.0
23	Hypertensive	30.0	24.0	20.0	
24	Hypertensive	33.2	34.8	No end point	*24.2
25	Rheumatic	No end point	No end point	No end point	No end point
26	Syphilitic	55.0	No end point	No end point	(H)50.0
27	Hypertensive arterio-sclerotic	No end point	No end point	No end point	No end point
28	Hypertensive arterio-sclerotic	23.0	23.0	No end point	(H)24.0
29	Hypertensive	47.6	45.6	No end point	47.0
30	Hypertensive	57.0	49.4	No end point	46.0
31	Rheumatic	24.2	19.0	18.2	
32	Rheumatic	No end point	No end point	No end point	28.0
33	Hypertensive	24.0	23.0	18.2	
34	Hypertensive	No end point	No end point	33.0	
35	Syphilitic	26.2	35.0	25.4	
36	Hypertensive arterio-sclerotic	23.0	21.0	No end point	No end point
37	Syphilitic	17.0	18.0	*10.0	
38	Coronary artery sclerosis	26.0	27.6	19.6	
39	Rheumatic	No end point	No end point	28.0	
40	Hypertensive	27.0	35.0	No end point	25.8
41	Hypertensive	26.8	24.0	No end point	22.0
42	Hypertensive arterio-sclerotic	30.2	25.6	*17.0	
43	Hypertensive	55.0	50.6	No end point	No end point
44	Hypertensive	20.0	16.8	No end point	(G)15.0
45	Coronary artery sclerosis	20.0	18.8	19.6	
46	Rheumatic	No end point	No end point	No end point	No end point
47	Hypertensive	30.0	32.0	28.0	
48	Coronary artery sclerosis	50.0	54.0	No end point	45.0
49	Rheumatic	20.0	21.0	18.0	
50	Hypertensive	19.4	20.6	19.0	

means of ether. Therefore, there were five cases (10 per cent) in which alpha lobeline failed to provide a record of the arm to tongue circulation time. Calcium gluconate failed to provoke any sensation in two cases (4 per cent). Magnesium sulfate was effective in all cases.

Measurements of the arm to tongue circulation time by means of alpha lobeline ranged from 8.2 seconds to 17.4 seconds (average, 12.6 seconds) in the 45 cases in which the drug gave an end point. There were 41 cases in which measurements obtained by means of alpha lobeline were shorter than those obtained with magnesium sulfate. In 13 of these cases the difference between the measurements was three seconds or more. The differences ranged from 0.2 second to 8.2 seconds, with an average difference of 2.2 seconds. There were 35 cases in which alpha lobeline gave a shorter measurement of the circulation time than calcium gluconate. In seven of these cases the difference was three seconds or more. The differences ranged from 0.2 second to 7.2 seconds (average, two seconds).

Measurements of the circulation time with calcium gluconate in the 48 cases in which the drug was used successfully ranged from 10 seconds to 18.6 seconds (average 13.8 seconds). There were five cases in which calcium gluconate gave a shorter measurement than alpha lobeline, but in only one case was the difference three seconds or more. The differences were from 0.2 second to 3.4 seconds (average 1.2 seconds). There were 29 cases in which calcium gluconate gave a shorter measurement than magnesium sulfate, but in only three cases was the difference three or more seconds. The differences ranged from 0.2 second to 4 seconds (average, 1.1 seconds).

Measurements of the arm to tongue circulation time with magnesium sulfate in the 50 cases ranged from 10 seconds to 20.8 seconds, with an average of 14.4 seconds. There were 10 cases in which magnesium sulfate gave shorter measurements than calcium gluconate. In none of these was the difference as great as 3 seconds. The differences were from 0.2 to 2.0 seconds (average, 0.9 second). There were two cases in which magnesium sulfate gave shorter measurements than alpha lobeline. The differences were 0.2 second and 0.8 second.

There were no alarming or unpleasant side effects from any of the drugs used in the 50 patients without heart failure. The cough which resulted from administration of alpha lobeline was short and mild. The sensation of warmth produced by calcium gluconate and magnesium sulfate passed away very quickly and was not disturbing.

In the 50 patients who had clinical evidence of heart failure, there were three cases (6 per cent) in which all three of the drugs failed to produce an end point. In the remaining 47 cases, the arm to tongue circulation time was found to be prolonged beyond the limits ordinarily accepted as normal. There were nine cases (18 per cent) in which calcium gluconate failed. There were also nine cases (18 per cent) in which magnesium sulfate failed. There were 27 cases in which 5 mg. of alpha lobeline failed to give an end point, but in only seven of these were 7.5 mg. of the drug ineffective. How-

ever, in five additional cases alpha lobeline gave a measurement of the arm to lung circulation time, so that there was a total of 12 cases (24 per cent) in which the drug failed as an agent for determination of the arm to tongue circulation time.

There were 26 cases of heart failure in which alpha lobeline gave shorter measurements of the arm to tongue circulation time than magnesium sulfate. In 17 of these cases the difference was three seconds or more. The differences ranged from 0.8 to 10.6 seconds (average, 4.6 seconds). There were 26 cases also in which alpha lobeline gave shorter measurements than calcium gluconate. In 16 of these cases the difference was three seconds or more, and the differences ranged from 0.4 to 11 seconds (average, 4.3 seconds). There were 26 cases in which magnesium sulfate yielded shorter measurements than calcium gluconate. Nine of these showed differences of three

TABLE III

Normal Circulation Time, Measured with Calcium Gluconate, Magnesium Sulfate, and Alpha Lobeline

Observers	Method	Range (Seconds)	Average (Seconds)
Goldberg [6]	Calcium gluconate	10–16	12.5
Baer and Slipakoff [14]	Calcium gluconate	9–16	12.7
Hussey, Cyr, and Katz	Calcium gluconate	10–18.6	13.8
Bernstein and Simkins [7]	Magnesium sulfate	7–17.8	12.9
Hussey, Cyr, and Katz	Magnesium sulfate	10–20.8	14.4
Berliner [10]	Alpha lobeline	5–18.0	9.3
Hussey, Cyr, and Katz	Alpha lobeline	8.2–17.4	12.6

or more seconds. The differences varied from 0.2 to 7.6 seconds (average, three seconds). In seven cases magnesium sulfate gave shorter measurements than alpha lobeline, and in four of these cases the difference was three seconds or more. The differences ranged from 1 to 13.2 seconds (average, 4.2 seconds). There were 17 cases in which calcium gluconate gave shorter measurements than magnesium sulfate, and in seven of these the difference was three seconds or more. Differences varied from 1 to 12.6 seconds (average, 3.9 seconds). In seven cases calcium gluconate gave shorter measurements than alpha lobeline, and in five of these the difference was three seconds or more. The range for the differences was from 1 to 25.8 seconds (average, 7.5 seconds).

As in the cases without heart failure, there were no disagreeable or dangerous side effects from the use of any of these drugs in patients with heart failure. As a matter of fact, some of the patients firmly believed that the administration of the drugs made them more comfortable.

DISCUSSION

From the studies reported here, it seems safe to conclude that the three drugs employed have an approximately equal usefulness in measurement of the arm to tongue circulation time. In the 50 patients who had no ap-

parent cause for a reduction in the velocity of blood flow, the ranges for the arm to tongue circulation time correspond closely with reports of other authors (table 3). The range for alpha lobeline is somewhat lower and the average arm to tongue circulation time is somewhat shorter than similar figures for magnesium sulfate and calcium gluconate. Furthermore, the circulation time measurements in the majority of the patients with heart failure were shorter with alpha lobeline than with either of the other two drugs. On the other hand, calcium gluconate and magnesium sulfate gave almost equal results in the two groups of cases. Therefore, when alpha lobeline is employed in clinical practice, it is necessary to remember that the results of measurement of the circulation time cannot be interpreted strictly in the light of previous experience with other agents, such as calcium, magnesium, and probably other compounds.

When any drug is used for the purpose of measuring the arm to tongue circulation time, failure of the drug to produce an end point is most commonly due to the fact that an adequate concentration does not reach the anatomical area of reaction or perception at any one time. This kind of failure may be due to the presence of a shunt which diverts the drug from the area of reaction, or to a lessening of the velocity of blood flow to such degree that the drug is too thoroughly diluted with the blood by the time it reaches the point of reaction, or to errors in the technic of injection, such as slowness of injection and failure of intravenous injection, or to the use of an insufficient dose. With regard to the matter of dosage of any of the agents used in measurement of the circulation time, certain standard doses have been recommended, more or less empirically. In some instances, dosage is limited because of toxicity, but the toxic limits for all drugs that have been used have not been determined as scientifically as might be desired. Ideally, the standard dose for each drug will be that amount which can be used safely and which will give a minimum of failures. Some such standard dose is necessary if measurement of the circulation time is to have value as an aid to clinical diagnosis for those who have not had wide experience in the use of the test. As experience is gained, it becomes possible to obtain accurate measurements while varying the dose of the drug employed according to the type of case. For example, measurement of the circulation time in a normal person by means of 20 per cent calcium gluconate will be the same with 2 or 3 c.c. of the drug as with 5 c.c. However, in a patient who has any cause for slowing of the circulation time, discrepancies will result from such variation in the dose of the drug. As a general rule, the use of larger amounts of any of these drugs will result in slightly lower figures for the circulation time. Further, when a specific amount of a drug is employed in a given patient, this same amount should be used later if the test is to be repeated. Otherwise, there will be no reliable basis for comparison of results. Other causes for failure of a drug used for measurement of the circulation time include insensitivity of the patient to the drug and failure or inability of the patient to coöperate during the test. In our series of 100 cases, the

percentage of failure was highest with alpha lobeline (17 per cent), next highest with calcium gluconate (11 per cent), and lowest with magnesium sulfate (9 per cent). Thirty of the 37 failures occurred in the patients with heart failure. In cases in which one drug fails to provide an end point for measurement of the circulation time, it is desirable to employ another drug. Thus, in only three of the 100 patients in our series did all three of the drugs fail. This kind of occurrence probably will be limited to cases in which there is extreme slowing of the circulation.

Alpha lobeline has one important advantage over the other two drugs used in our study. This is the fact that it gives an objective end point. It has, therefore, a unique field of usefulness in uncoöperative patients and in patients who are unconscious or stuporous. Its value is enhanced by the knowledge that a grimace is sometimes the only indication of its end point. In our experience, hyperpnea is not reliable as an end point. This phenomenon can be observed in practically all of the cases in which the drug is used, but many seconds after the appearance of a grimace or cough. Hence, in cases in which hyperpnea is the only reaction to administration of the drug, it is safe to assume that the drug reached the carotid sinus long before the reaction appears. This assumption is supported in such cases by the marked discrepancy between measurement of the circulation time by means of alpha lobeline and that obtained with calcium gluconate or magnesium sulfate. Apparently the only objections to the use of alpha lobeline for determination of the arm to tongue circulation time are that it often fails in a dose of 7.5 mg., and it occasionally provides a measurement of the arm to lung circulation time. The first objection probably can be solved in part by the use of larger doses. Probably 7.5 mg. are always safe as an initial dose for adults of average size, and doses up to 12 mg. have been used.[10] The second objection is apparently insurmountable and may cause considerable confusion in interpretation of results unless they are verified routinely by means of another drug. We have seen no unpleasant effects from the administration of alpha lobeline. Hemoptysis has been mentioned as the only definite contraindication to the drug.[10] However, two patients in our series who were having hemoptysis were not adversely affected by alpha lobeline, nor were any of the patients with acute or chronic inflammatory conditions in the lungs. We have not found any necessity for a delay between injections when the use of the drug is repeated in the same patient.

From a study of the statistics in tables 1 and 2, it seems safe to say that calcium gluconate and magnesium sulfate give results which are equally satisfactory for measurement of the arm to tongue circulation time. Neither has an unpleasant effect, and apparently neither is dangerous in the doses recommended. However, a theoretical objection has been raised to the use of calcium in patients receiving digitalis.[13] This objection is based on the knowledge that calcium and digitalis have an additive effect on the heart. Nevertheless, the dose of calcium gluconate recommended for measurement of the circulation time seems to be entirely without danger.

Summary and Conclusions

1. The results of measurement of the arm to tongue circulation time with alpha lobeline, calcium gluconate, and magnesium sulfate have been compared in 50 patients without cause for slowing of the circulation and in 50 patients with heart failure.

2. Alpha lobeline gave somewhat shorter measurements than calcium gluconate and magnesium sulfate in both groups of cases, but failed to provide an end point more often than either of the other drugs. Furthermore, the fact that it sometimes measured the arm to lung circulation time seems to detract from its value for routine use. Its chief advantage consists in the objectivity of its end point.

3. Calcium gluconate and magnesium sulfate gave approximately equal results. They seem to be equally valuable for routine use in measurement of the arm to tongue circulation time, and perhaps are more valuable in this respect than alpha lobeline.

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CASE REPORTS

CALCINOSIS TREATED BY PARATHYROIDECTOMY *

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Subcutaneous calcinosis, a disease tending to serious bodily disability, presents a most difficult therapeutic problem. It is usually the sequel to dermatomyositis or some inflammatory disease in the skin or subcutaneous tissue, and produces difficulty by interfering with proper skeletal function by restricting the movements of muscles and tendons. The circulation of the skin and surrounding tissues may be disturbed and, at times, ulceration and infection may take place in the region of the calcium deposits.

Since the calcium deposits are usually considered to be fixed and the destruction of muscles and tendons irreparable, any type of treatment seems almost doomed to failure. Acid salts, such as ammonium chloride, have been used in an attempt to draw calcium from its deposits into the blood stream where it can be excreted normally. This method seems rational, but the results are usually disappointing. The production of ketosis, another method of attacking calcium stores, was reported to be successful in a case by Kennedy.

Ramsdell reported striking results in a young girl with calcinosis, following the production of tetany induced by unilateral parathyroidectomy. This plan of treatment likewise has as its motive the mobilization of calcium. It was utilized in the case herein reported.

Case Report

A boy of 14 was first seen at the Lahey Clinic in October 1938, because of tenderness and swelling of the right wrist, which had been present for five days. A long illness had antedated the development of the recent acute condition. At the age of eight, following an injury to his face while riding on a sled, he noted weakness and stiffness in the extremities and discoloration of the skin. A diagnosis of dermatomyositis was made at that time, and physiotherapy was advised. Although the boy was able to be up and around, his muscular activity became greatly reduced so that he could not kneel or completely extend his arms. As time went on, white hard material was deposited in the skin and subcutaneous tissue over the arms, legs and trunk. The skin over these deposits occasionally broke, and thick, chalky material drained. It was in the region of one of these deposits that the tenderness and swelling developed which brought the boy to the clinic.

Physical examination showed that the patient was fairly well developed, but moderately undernourished (figure 1). He weighed 98 pounds. The pulse and blood pressure were within normal limits. The skin of the face was mottled, there being linear regions of thickening and atrophy, with some capillary dilatation. The skin over the arms, legs and trunk was slightly erythematous and thickened, with fine scaling. Numerous white superficial nodules, measuring from 2 mm. to 1 cm. in

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diameter, were present over the arms and legs, and somewhat larger nodules were found over both elbows. The right wrist was red, swollen and intensely tender. The muscles of the forearms and legs were firm and markedly atrophied. The forearms could not be extended beyond a 60 degree angle, nor could the lower leg be flexed on the thigh, the patient thus being unable to kneel. The fingertips and ends of the toes contained superficial plaques similar to those on the forearm. Capillary dilatation was present in the region of the cuticle of the fingernails and toenails.

Examination of the blood revealed the hemoglobin to be 96 per cent; erythrocytes numbered 5,150,000 and leukocytes, 10,300. Urinalysis and the Hinton test gave negative results. The serum calcium, phosphorus and phosphate determinations were all within normal limits.

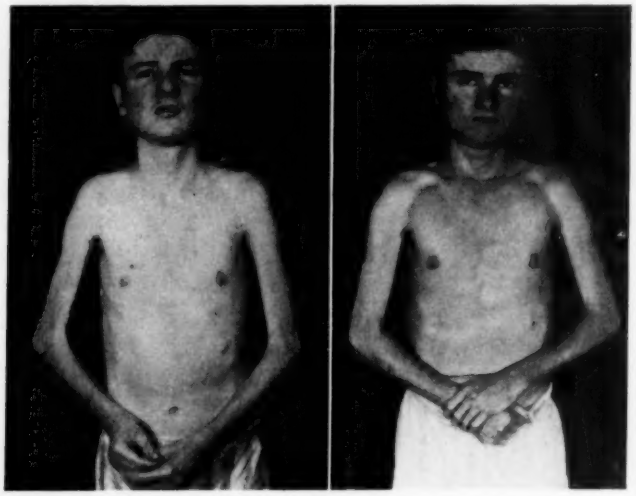

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FIG. 1. Improvement in general physical condition may be noted. Arms can be flexed to a greater degree than when the patient first came to the clinic.

Roentgenograms of the chest, extremities and abdomen demonstrated multiple, scattered, irregular areas of calcification in the subcutaneous tissue (figure 2). The calcium content of the bone appeared normal.

A specimen of skin and subcutaneous tissue was removed from the region of the right scapula for microscopic study. Localized fibrosis with chronic inflammation and calcification was found.

A diagnosis was made of calcinosis secondary to dermatomyositis, acrodermatitis chronica atrophicans and cellulitis of the right wrist, resulting from infection in a calcium deposit. The infection in the region of the wrist subsided promptly following treatment with continuous warm packs. Treatment aimed at the general disease was then considered. Because of the benefit from a ketogenic diet, reported by Kennedy, the patient was placed on this diet and, in addition, was given 45 grains of ammonium chloride and ½ grain of Armour's desiccated thyroid each day. Lanolin was used locally on the skin.

The patient's course was followed for eight months on this plan of treatment. It was extremely difficult to keep him in ketosis as he did not coöperate well in continuing his diet and took ammonium chloride only periodically. During this time two infections, one on the left foot and one on the elbow, developed, causing considerable

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FIG. 2. Note absorption of large calcium plaque on anterior aspect of the forearm.

pain. In both instances a large amount of chalky substance drained from the infected areas before healing took place. The patient gained seven pounds, but no change took place in the skin or function of the extremities.

Because of the lack of discernible improvement on this regimen, it was decided to employ other therapy. It was at this time that Ramsdell reported his success in a similar case following unilateral parathyroidectomy, and operation was advised for this patient.

Operation was performed August 14, 1939, under cyclopropane ether anesthesia with carbon dioxide absorption technic. Through the usual collar incision, the pre-

thyroid muscles were divided on the right side, exposing the right lobe of the thyroid. After division of the middle thyroid vein, the inferior parathyroid was demonstrated and totally removed. Frozen section demonstrated it to be parathyroid tissue. Following this, the superior pole of the right lobe was mobilized and several bodies suggestive of parathyroid glands were removed. None showed parathyroid tissue. In order to complete the dissection, the upper third of the right lobe of the thyroid was removed. Both superior and inferior thyroid arteries were ligated. The left lobe of the thyroid was not disturbed.

The patient's postoperative course was satisfactory. Although a positive Chvostek sign was elicited after the operation, no subjective symptoms of tetany de-

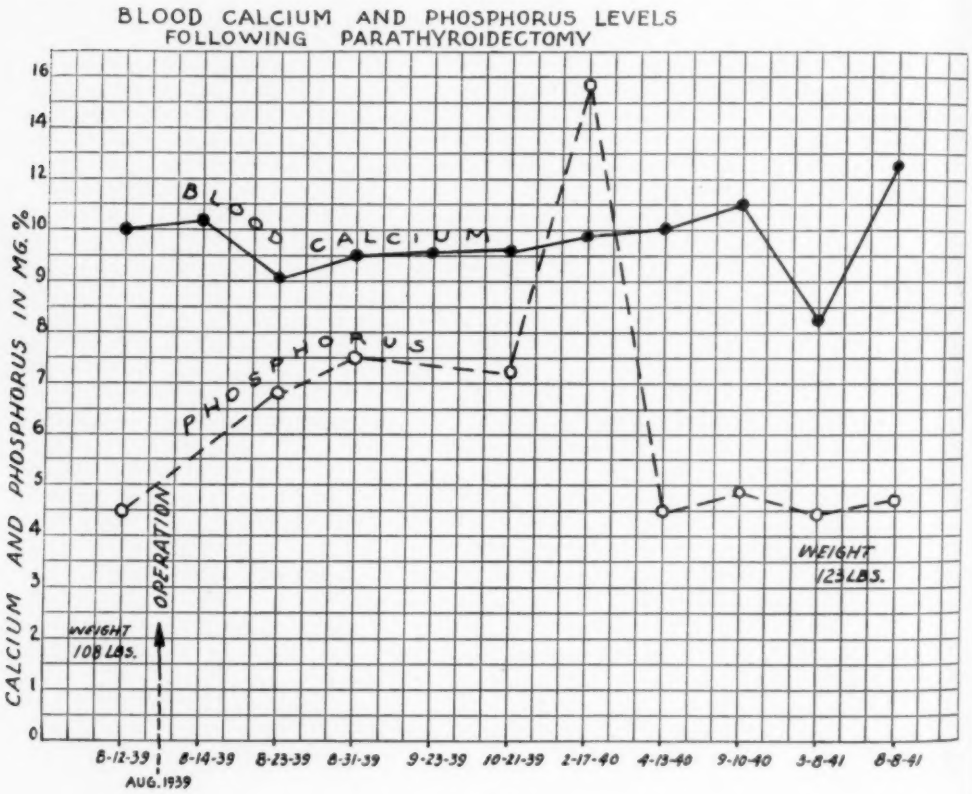

FIG. 3.

veloped. He was discharged from the hospital five days after operation. The subsequent level of the serum calcium and phosphorus is shown in figure 3. There was a steady gain in weight after the operation so that at the end of eight weeks he had gained seven pounds. Soon after the operation, noticeable loosening of the extremities took place.

Two years have now elapsed since the operation. The patient's general health has markedly improved; he has gained 15 pounds in weight (figure 1). All ulcerated areas have healed, the texture of the skin has improved, and many of the calcium deposits have disappeared. The function of the arms has improved, and the patient can now kneel with ease. The muscles have increased in size and strength. A roent-

genogram (figure 2) shows almost complete absorption of one large calcium deposit on the anterior surface of the forearm. A few deposits are still visible on the posterior aspect.

Summary and Conclusions

A boy of 14 was observed who had calcinosis of the subcutaneous tissue and muscles, associated with acrodermatitis chronica atrophicans secondary to dermatomyositis. When the patient was first seen there was marked restriction of the motion of the extremities and infection in the region of the calcium deposits. Treatment with a ketogenic diet, ammonium chloride and desiccated thyroid for one year produced no apparent improvement. At the end of this time, unilateral parathyroidectomy was performed, producing chronic tetany. No subjective symptoms of tetany developed, although a positive Chvostek sign was elicited. The serum phosphorus level became elevated and remained so for a period of eight months; the value for the serum calcium remained normal. During the two years since operation marked improvement has occurred, and roentgenograms demonstrate absorption of some of the calcium deposits. All the skin infections have healed, and the texture of the skin has improved.

The response of the serum calcium and phosphorus in this patient following parathyroidectomy suggests that the parathyroid glands are primarily involved with phosphorus excretion, and therefore account for the increase in the serum phosphorus level. The failure of the serum calcium level to fall may be explained on the basis that the mobilization of calcium from the calcium deposits in the subcutaneous tissue replaces that lost through urinary excretion. As the blood elements have returned to normal, no further clinical improvement is now anticipated.

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MULTIPLE MYELOMA*

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Multiple myeloma is considered a malignant tumor exhibiting multiple foci throughout the red bone marrow in the body. One of the first cases of this disease reported in 1845 by William McIntyre received very little attention from the medical profession. Not until 1882 was it definitely recognized after presentation by Kuhne and it was further described in 1889 by Kahler. The etiology of the disease is as yet unknown. The majority of cases occur in the male between the fourth and sixth decades. In the cases reported, the principal

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symptoms have been pain, tumor, deformity, and fracture, and there may be systemic changes affecting the respiratory and cardiorenal systems and at times the gastrointestinal tract and blood stream.

The pain is frequently intermittent and is generally referred to the back and to the extremities. It is aggravated by movement and increases in intensity as the disease progresses. Periods of remission are not uncommon.

The presence of small tumors distributed over the ribs, sternum, clavicles, spine and extremities are important diagnostic criteria when present. They may

FIG. 1. (*Above*) The rounded head of the femur with collapse of the entire upper end of the femur. (*Below*) Tibia. This section reveals the diffuse character of the hemorrhage into the tumor and its distention, with thinning of the cortical layer of the shaft.

result in pathological fractures. Neurological manifestations, if present, are the result of compression of the cord by these tumors and may result in paralysis, and in bladder and rectal incontinence which in turn may lead to decubitus ulcers. Kidney changes are not characteristic, but albuminuria is frequently present and the albumin-globulin ratio may be reversed. The blood picture is not characteristic except for a marked anemia. Roentgenologic study of the bones forms the most important diagnostic procedure, although the final diagnosis must be made by microscopic study.

CASE REPORT

This report is of a white male, 25 years of age, who was admitted to the hospital November 13, 1936. He gave a history of having been apparently well until September 1936, at which time he first noticed the development of a dull, ache-like discomfort in

both thighs which felt deep as though it were in the bones, and which were present more often when he was tired. Believing this discomfort to be merely the result of over-exertion, he did nothing about it until August 1937 at which time he went to a chiropractor for therapy, and two months of chiropractic treatment consisting of massage and light did not help to any great extent. From September 1937 until July 1938 he had no treatment at all, but in July 1938 he again began to have severe steady aches which developed in both hip regions and in the lumbosacral spine, and again he sought medical advice. Physiotherapeutic measures were tried until October at which time the pain became more severe and he was unable to continue his work as a steel catcher. The pain was aggravated by bending over or lifting heavy objects, and on occasions the pain was so severe that he fainted. At this same time the patient noted a change in the character of the pain. Whereas previously it had been dull, aching in character, deeply situated, now it became sharp, stabbing, and at times lancinating in character, aggravated by any movement of the lower spine or hips. It was not continuous, however, and when the patient lay perfectly quiet he was fairly comfortable. In October 1938 he found it necessary to use a cane when walking and even then experienced such severe pain in the lower spine, thighs and hips that absolute bed rest was advised and he was referred to the Medical Service for further care.

The patient had had no definite complaints referable to any of the systems of the body other than those already mentioned in reference to his locomotor activity. He had never used alcohol and had been free of venereal disease. He had had the usual childhood diseases. He had had a fracture of the right humerus at 11 years of age, and a fracture of the right ankle at 17 years of age.

The family history was negative for any chronic disease, and his father, mother and 10 siblings were alive and well. He was married, but had no children.

Upon admission to the ward the patient appeared to be a well developed, poorly nourished white male of 25 years, not in acute pain while lying in bed, but expressing a great deal of pain on any attempt at motion for examination. Essential physical findings were as follows: Head: There was no evidence of enlargement, depressions or previous injury. Eyes: The pupils were round, equal, regular, reacted to light and on accommodation. The conjunctivae appeared pale. Fundi were negative. Ears: Both drums were clear with a normal light reflex. Nose: The septum deviated slightly to the right. There were no mucous membrane changes. Mouth: The lips were pale; the tongue protruded in the midline; the mucous membranes were clear. The neck was flaccid; the cervical glands were not palpable. The trachea was in the midline. The chest was equal and symmetrical; fremitus and resonance were not impaired. The breath sounds were vesicular in quality and came through well in all portions of the lungs. The heart: The apex was in the fifth interspace, 8.5 cm. from the midsternal line. The base was not enlarged to percussion. The heart sounds were regular and of good tone; there were no murmurs. The radial vessels were moderately sclerotic. Blood pressure was 160 mm. Hg systolic and 100 mm. diastolic. The abdomen was soft and not tender. The liver and spleen were not enlarged. There was no spasm, and no masses were palpable. The genitalia were those of a normal male. The spine was held rigid. There was kyphosis of the lumbar region but no evidences of tumor masses. There were no tender spots elicited on palpation of the spine. Extremities showed no edema.

On admission to the ward the red blood count was 2,290,000 with 56 per cent hemoglobin. The leukocyte count was 9,400, with 49 per cent lymphocytes, 2 per cent monocytes, the remainder in the polymorphonuclear series. The red cells showed moderate variation in size and shape. Three nucleated red cells were noted; diffuse basophilia was present. Color index was 0.9. Subsequent counts showed much variation, between 2,300,000 and 3,200,000 red cells and 4,000 to 9,000 leukocytes. The differential count varied, but in most instances was normal with an

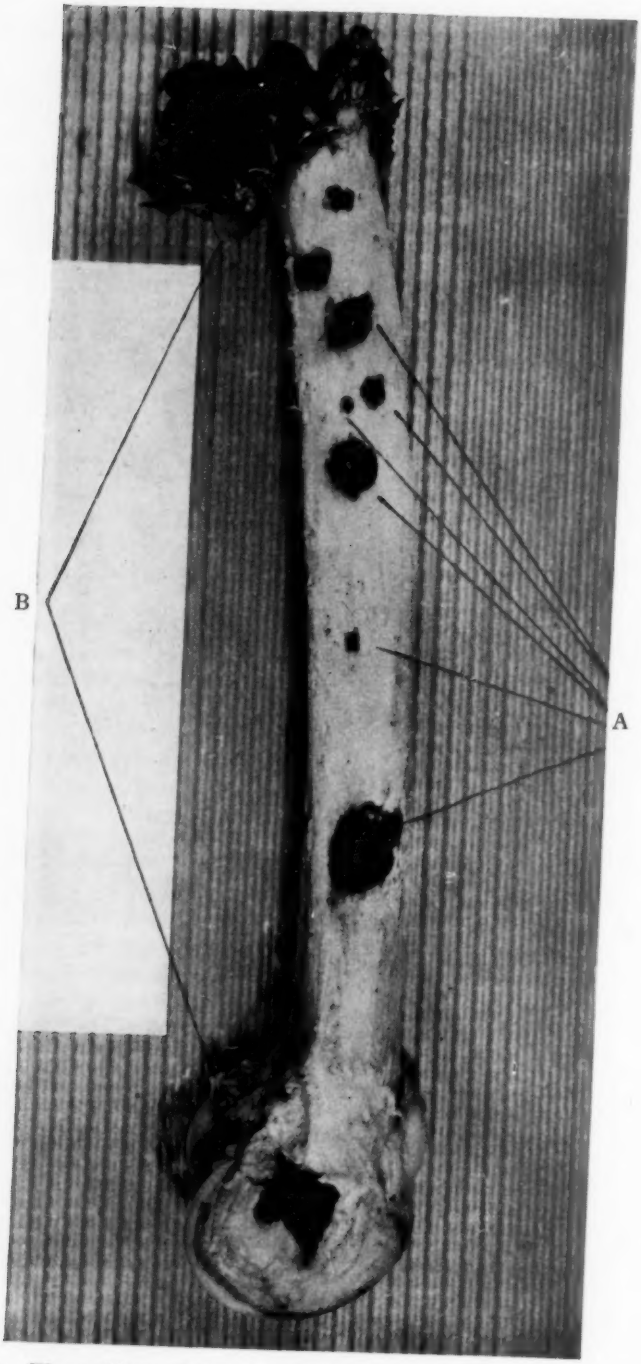

FIG. 2. Femur. The outlines of the circumscribed tumor nodules, *A*, can be seen on the cortical surface. The thin shell-like character is noted at both ends, *B*.

occasional relative lymphocytosis. The red cell count was typical of a progressive chronic disease and although at first it showed definite improvement on iron and liver therapy, it soon began to show a gradual decrease in spite of the therapy offered. Approximately one year following his admission his count had dropped to 1,660,000 with 38 per cent hemoglobin and there was a leukopenia of 2,350 with 36 per cent lymphocytes, and 64 per cent polymorphonuclears.

Subsequently only slight and transient improvement was obtained by repeated transfusions, and on March 16, 1940, the red cell count had fallen to 900,000, with 23 per cent, or 3.4 grams of hemoglobin.

Urinalysis done approximately weekly during the patient's stay in the hospital revealed occasionally a trace of albumin or sugar, but as a general rule was negative. Specific gravity varied from 1.010 to 1.015 with an adequate urinary output. The sediment on most occasions was normal. Tests for Bence-Jones protein on 15 occasions were uniformly negative.

Blood chemistry revealed the following data. The blood urea was 12 mg. per 100 c.c. on admission, rose to 18 mg. in February 1939, and remained at approximately this figure except for a terminal elevation to 22 mg. per 100 c.c. The blood sugar varied between 82 and 96 mg. Kidney function tests showed a moderately low specific gravity with satisfactory urinary output as evidenced by the Mosenthal test, whereas phenolsulfonphthalein tests showed kidney impairment with excretion between 45 per cent and 55 per cent of the dye in two hours. On November 20, approximately one week following his admission, the blood calcium level was 13 mg., the inorganic phosphorus 4.3 mg., total proteins were 6.55, albumin 2.82, globulin 3.73, and albumin-globulin ratio 0.72. Phosphatase at this time was 3.2 Bodansky units and ionized calcium, 6.2 mg. The total protein a month after his admission was essentially the same with an albumin-globulin ratio of .76. In November of 1939 the blood calcium was 13.17 mg., the inorganic phosphorus 5.2 mg.; approximately three months later the blood calcium was 12.51 mg., the inorganic phosphorus 4.9 mg. In April 1939 the blood calcium was 13 mg., and phosphorus was 4.8 mg. Sedimentation rate done on four occasions showed an approximate average of 14 mm. Blood cholesterol was 174 mg., and blood chlorides 630 mg.

On November 19, 1939 a roentgenogram of the chest showed the lung fields to be clear. The heart and aorta were within normal limits, the diaphragms normal in position and contour. The spine, particularly the lumbar, all pelvic bones, both femurs and humeri, all ribs, scapulae and the skull showed numerous punched out, sharply delineated areas of decreased density, typical of multiple myeloma. Because of this, a rib biopsy was done on December 6, 1938, the material consisting of a fragment of rib 4 cm. in length which, when split through, showed a clear, gelatinous-like material in a sharply punched out area. When sectioned these areas showed a profuse proliferation of cells with occasional mitotic figures, that were larger than the small lymphocyte, more nearly approximating a large lymphocyte. Some of these cells showed reticulation of their nuclei; some showed cytoplasmic processes which made a delicate reticulum in which these cells were found. There were also small clusters scattered in and among the cells that were definitely small lymphocytes. None of the normal bone marrow elements could be made out. Occasionally there was a large cell resembling a megakaryocyte. The diagnosis was multiple myeloma.

Roentgen-ray examination repeated in January 1939 revealed a definite progression of the myelomatous lesions in the skull, lumbar spine, pelvic bones and along the shafts of the femur. The forearms, legs, hands and feet showed no evidence of lesions. Roentgenogram of the chest, repeated because of chest complaints, revealed the lung fields clear with no evidence of lung metastasis. In March 1939 again roentgenograms showed marked progression of the myelomatous lesions in the pelvic bones, upper thirds of the femurs, lumbar spine, some in the ribs, skull and humeri.

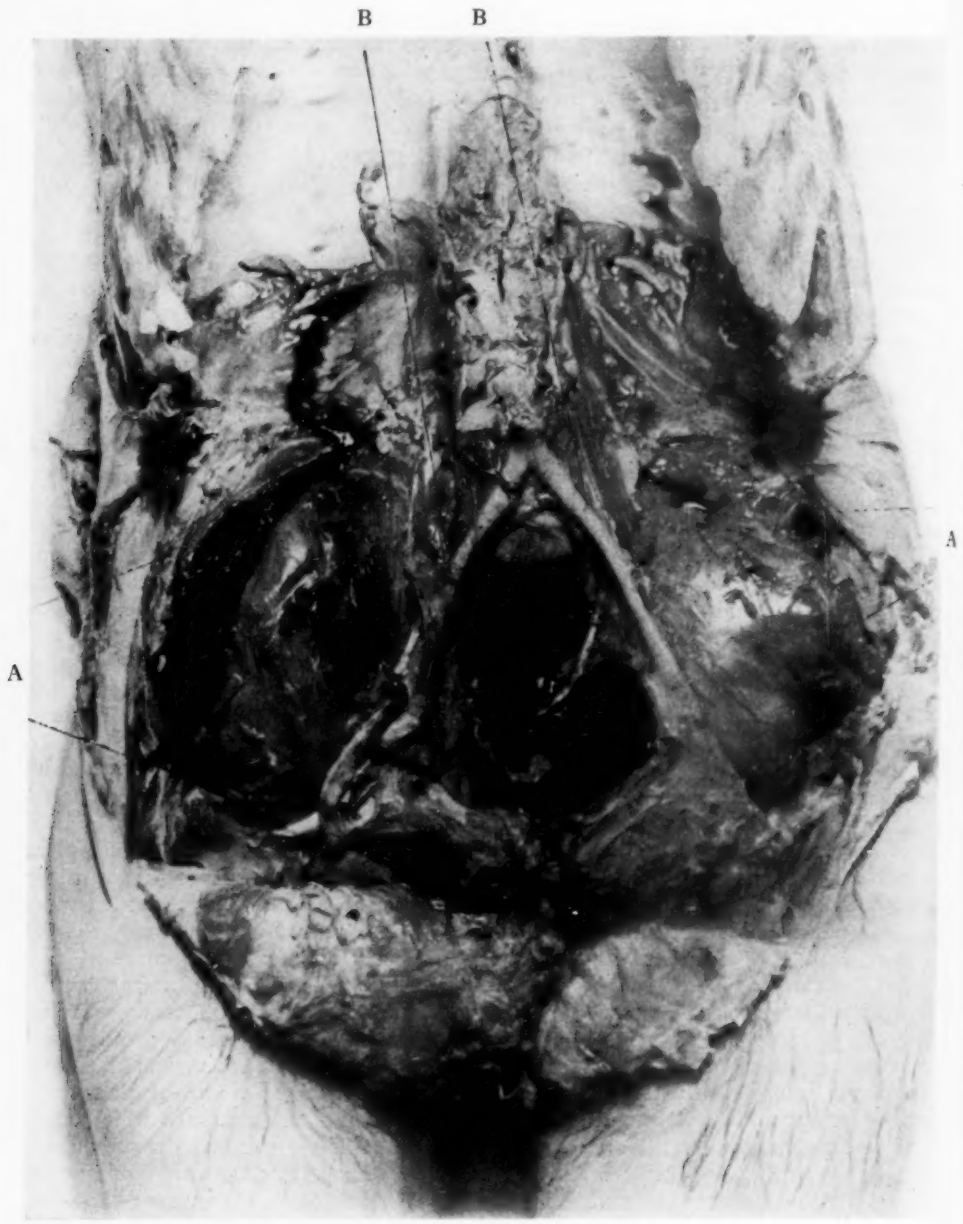

FIG. 3. The pelvic picture reveals two large bulges, *A*, one on each side, outlined mesially by the bifurcated aorta, *B*. The mass on the right side has been incised to reveal the diffuse hemorrhagic character of the tumor. The psoas muscle and sciatic nerves as well as the returning veins are stretched and compressed by the bulging. The osseous structure was reduced to sand-like remains.

The course of the case during his stay in the hospital from the time of admission until March 23, 1940 was that of a chronic progressive fatal disease. Because of the severe pain the patient was placed on a hard board mattress but found little relief and was kept as comfortable as possible with narcotics. Therapy for the anemia has already been mentioned and other therapy was purely symptomatic.

In February 1939 he began to exhibit frequency, urgency and incontinence of urine, following which a diagnosis was made of neurogenic dysfunction of the bladder. Antispasmodics were given with some relief.

Dermatologically two complications developed in the course of the disease. In March 1937 herpes zoster was noted, which improved satisfactorily on salicylate treatment and calamine lotion. Two years later there was noted on each shoulder

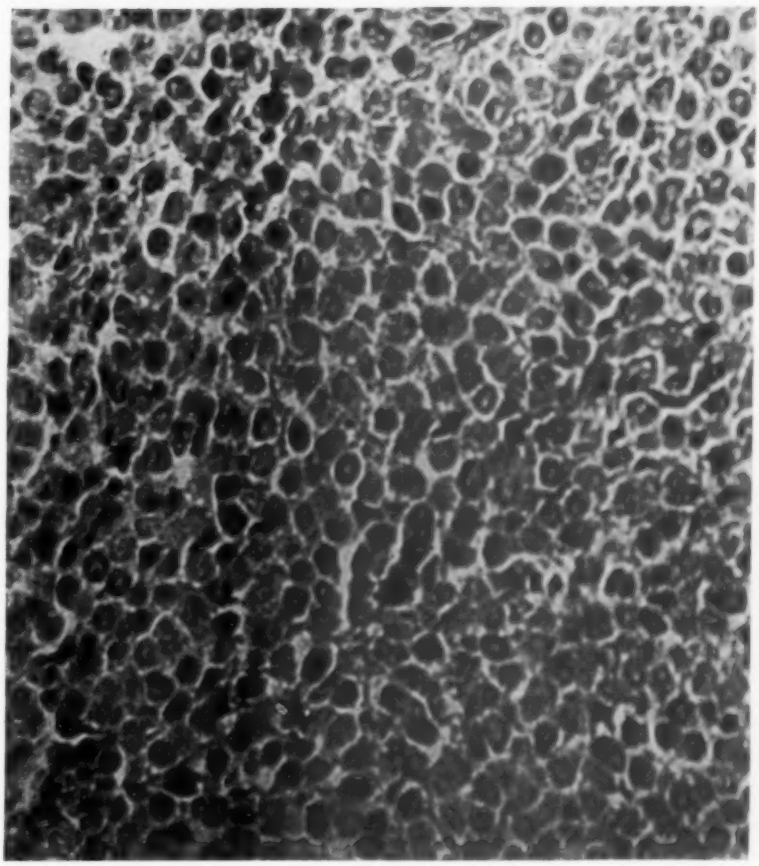

FIG. 4. The cell type is not clearly the plasma cell; the pyknotic nucleus appears only at times, then only suggestively, the cytoplasm generally is basophilic, clear or fragmented. The mononuclear cell with definite nuclear membrane, narrow rim of cytoplasm is the more common type. Mitotic figures are few. Diffuse hemorrhage with fibrin dominates the picture; many nucleated reds are present. The breakdown of the osseous trabecula is complete and extends into the cortical layers which are reduced to a thin shell. The latter have been perforated by periosteal invasion that presents either as diffuse masses or extensive infiltrates, e.g. the ribs which are 2½ times the normal diameter. Or the thin cortical layers are tremendously distended with hemorrhage and tumor as in the ilia.

FIG. 5. (*Above*) Roentgenogram taken three days after admission to the hospital, estimated to be 18 to 20 months after the onset of the disease. Note the characteristic rounded punched out areas with their evident predilection for the cancellous structures. Even now confluence is in evidence throughout the involved fields. (*Below*) The marked progression of the disease is strikingly demonstrated by comparison with the cut above. Pathological fractures of the pelvis and both hips are now present. Notwithstanding the great increase in size of the lesions, a circumscribed outline is easily seen and helps to differentiate from carcinomatosis.

FIG. 6. Owing to the numerous scattered lesions of myeloma the bodies of the first, third, fourth and fifth lumbar vertebrae are compressed. There is also narrowing of the intervertebral spaces, particularly between the third and fourth vertebrae.

posteriorly a palm-sized plaque of infiltration of the cutis, which was confined to the dermis and was moderately tender on pressure and diagnosed as calcinosis cutis.

In October 1939, approximately a year following his admission, he began to show edema of his left leg, which gradually extended upward to involve the penis and scrotum. This seemed to subside somewhat but he never definitely recovered.

In March 1940 his condition became more serious. He began to complain of dyspnea; his respirations were rapid; and his lungs showed bilateral basilar congestion. His heart sounds were poor, the pulse was rapid with a rate of 120 per minute, but no definite bruits were heard. He complained of generalized pain throughout his body.

He died on March 21, 1940 with a final diagnosis of multiple myeloma, marked hypochromic anemia, and circulatory failure.

Postmortem examination was made on March 22, 1940. The body was moderately well developed and nourished, but the extremities, especially the lower ones, lacked tone, were flaccid and semifluctuant. The skin surface was shiny. There was an increase in the lateral diameter of all extremities. The bones of the knee and hip joints were friable and compressible with a fine crackling crepitus on palpation. The soft tissues were edematous. Postmortem lividity and rigidity were present.

The ribs were mottled and carious and there were soft reddish bulges from the softened areas. The pleura was smooth and glistening. The lungs were voluminous, grayish-pink and dark gray. Crepitus was absent throughout. Cut sections showed the same mixed coloration with a marked pouring out of thin serous froth from the cut surface.

The pericardium was smooth and glistening; the heart was normal in size; the muscle was pale and flabby. The valves and orifices were clear. The left ventricle was three times as thick as the right. Coronary vessels were patent. The aorta was thin and showed wrinkling and yellowish plaques on its intimal surface.

Gastrointestinal: The esophagus was not remarkable. The pylorus was contracted, the duodenum small, the large intestine not remarkable.

The liver was slightly reduced in size with a smooth capsule. The spleen was approximately three times normal in size, its color diffuse bloody red. The adrenal glands were of normal size. The gall-bladder was thin walled and contained 20 c.c. of dark inspissated bile.

Both kidneys were slightly smaller than normal; the capsule was thin and stripped easily, revealing a smooth surface. In the cortical portion in the columns of Bertini there were noted slight streakings, following especially the lines of the straight tubules, which proved to be calcium deposits due to a high blood calcium during his life. The pelves and ureters were clear, the bladder small. The prostate gland was normal in size.

Skeletal system: All the bones were involved to a lesser or greater degree by the tumor, especially the long bones. On opening the left femur the left hip joint was found to be completely disintegrated, and the myeloma had destroyed the osseous structure and invaded the soft tissues. Similarly, in the pelvis, both ilia were massive, larger than grapefruit size, bulging forward and upward, compressing the vessels and the lumbosacral nerves, so that venous circulation was retarded, resulting in edema. These masses on dissection were hemorrhagic, consisting of fat, and bony spicules, being completely disintegrated. The remainder of the femur showed circumscribed areas varying in size. The lower end of the femur and the upper end of the tibia were completely broken down. Both tibiae showed mottling and points of perforation by the myeloma. The vertebral column was similarly involved. Permission to examine the cranium was not obtained.

The skin showed circumscribed, hard, pale plaques which on section proved to contain calcium.

The final anatomical diagnosis, which was in accord with the clinical impression, was: (1) Multiple myeloma with hemorrhages, bone absorption and multiple fractures; (2) marked secondary anemia; (3) metastatic calcification of kidneys and skin; (4) myocardial degeneration and dilatation; (5) pulmonary edema.

Summary

It is of interest that although red cell regeneration was active, the anemia was pronounced. Another feature of interest was the complete loss of osseous structural support that exists in the normal skeletal frame. The body as a

whole seemed like a fluctuant mass, flattened and collapsed; each handling was productive of further disruption of continuity of the osseous structure. Another point of interest was the edema of the lower extremities, which was readily accounted for by the obstruction to the venous return produced by the pelvic masses.

We have, then, a case of multiple myeloma of approximately four years' duration observed in a young male 25 years of age. He shows the characteristic history of vague pains gradually becoming more severe, and involving chiefly the lower spine and lower extremities. The laboratory data in this case were, for the most part, in accord with the usual findings, except that Bence-Jones protein could not be demonstrated in the urine although examined on approximately 15 occasions. A high blood pressure was repeatedly observed, as a result of calcium deposit in the kidney. The clinical course and laboratory data were in accord with those usually reported in this disease.

We are indebted to Dr. William F. Jacobs, pathologist of the Edward J. Meyer Memorial Hospital, for the pathologic report in this case, and to Dr. Clifford R. Orr for the roentgenologic studies.

EDITORIAL

THE WAR AND MEDICAL EDUCATION

ALTHOUGH our country has been at war less than a year, extensive changes in medical education have already been instituted in order to meet special conditions created by the immediate needs of our armed forces. These changes are both quantitative and qualitative in character.

The principal quantitative change that has been put into effect is the adoption by the majority of medical schools of an accelerated program whereby the medical course is shortened from four to three calendar years. This means that the four academic years can now be completed within three calendar years by continuous instruction throughout the summer months at the sacrifice of the lengthy summer vacations so welcome to students and faculty alike in peacetime. There has been no reduction in the total number of hours devoted to instruction in the medical sciences—in other words, no lowering of the standards of the medical school or of the requirements for the degree of Doctor of Medicine. Since the majority of colleges that offer premedical courses have also adopted an accelerated program, it is now possible for a student to obtain the A.B. or B.S. degree in three calendar years and the M.D. degree in three more calendar years, a total of six years of undergraduate and professional education as against the eight years previously required for such an accomplishment.

As a result of the accelerated program, medical schools will graduate a full class every nine months. These graduates will be eligible for service with the army or navy medical corps as soon as they have completed a nine to twelve months interneship. Hospitals have changed their dates for interneships to dovetail with the graduation dates of the accelerated medical schools. Such a speeding up of medical education should prove an important factor in keeping our armed forces supplied with a steady stream of vigorous young medical officers, well prepared for active duty in each and every theater of the war.

The accelerated program has been subjected to certain unfavorable criticism. It is undoubtedly true, as many have alleged, that the students may be less alert and "grow stale" under the pressure of almost continuous instruction without benefit of a long summer vacation in which to "digest" the learning that they have imbibed during the previous academic year. This necessarily unfortunate state of affairs is more than offset by the sense of satisfaction on the part of the students that they are wasting no time in preparing themselves to "get in the thick of the fight." A good many medical students find it difficult to justify to themselves staying on in school when a large number of their non-professional contemporaries are already on active duty with the armed forces. Surely, it must be some consolation to them to

know that the Army and Navy regard our medical schools in much the same light as West Point and Annapolis, that is as training grounds for future officers.

To those medical students of limited means the accelerated program has presented an acute financial problem. Tuition bills must be met more frequently, yet there is no longer the opportunity to earn money during the summer months with which to help defray expenses. Loan and scholarship funds have been established by the United States Government and by the Kellogg Foundation in order to meet just such an exigency. The medical schools, themselves, can do their part by offering more scholarships, raising loan funds, and granting deferment of tuition in special cases.

Coincident with the adoption of the accelerated program, medical schools have been obliged to strip their faculties down to a skeleton force of "essential" instructors in order to release as many physicians, surgeons and scientific specialists as possible for service with the armed forces. Thus a relatively small staff must assume the responsibility of carrying the full load of the year-around teaching schedule and wartime research. In addition to the instruction of medical students, these depleted faculties will be expected to offer refresher courses, in subjects of military importance to medical officers of the Army and Navy. We may confidently expect that all those instructors who have been prevented from joining the armed forces of the nation, either through physical defects or through their "essentialness" to their medical schools, will shoulder these added teaching burdens with wholehearted enthusiasm as their contribution to the war effort. Full-time men must expect to work longer hours and to subjugate their own research interests to the immediate demands upon their time, while part-time instructors should and undoubtedly will devote a larger portion of their time to teaching in spite of the increase in their private practice resulting from the shortage of physicians remaining in civilian practice.

The Selective Service status of medical and premedical students raises a number of problems of considerable interest. Under the original Selective Service Act, a recommendation was issued to local draft boards that all medical students in good standing be classified II–A—that is, deferment for occupational reasons. More recently, medical students have been permitted to apply for reserve commissions as second lieutenant in the Medical Administrative Corps of the Army or as ensign (H. V. P.) in the Medical Reserve Corps of the Navy. Such a tentative appointment protects a medical student from being drafted throughout his medical school career and a one-year interneship, after the completion of which he is subject to call for active duty as first lieutenant in the Army Medical Corps or lieutenant (j. g.) in the Navy Medical Corps. Medical schools have been requested not to ask draft deferment for students who have failed to make application for such a reserve commission. Premedical students who have been admitted to a Grade A medical school may apply for similar commissions, provided that they expect to enter medical school within one year. The opportunities afforded

by such legislation have served to prevent all medical students and the majority of premedical students from being drafted up to the present time. However, if the lower draft age limit is dropped to 18 years, it is obvious that further measures should be forthcoming. We could scarcely expect the medical schools of the country to admit students on the basis of their accomplishments in high school or during their freshman year at college. Government provision would have to be made for protecting bona fide premedical students from being drafted, possibly on the basis of a certificate from the dean of the college. Otherwise, medical schools would be obliged to close their doors for lack of a student body.

Qualitative changes in the curriculum of medical schools have been designed to emphasize in particular traumatic surgery, diseases of young adults, and infectious diseases prevailing in the various countries and climates to which our armed forces may be exposed. Every medical student should become thoroughly familiar with such infections as influenza, pneumonia, meningitis, rheumatic fever, syphilis, gonorrhea, chancroid, the dysenteries, enteric fever, typhus, malaria, and tropical diseases rarely encountered in this country such as yellow fever, kala-azar, dengue, Australian "Q" disease, and a variety of others. He must be drilled in the proper technic of debridement of wounds, the treatment of burns and shock, and chemotherapy with the sulfonamide drugs. In order to attain such a goal, additional time must be allotted for practical training of the students in the accident room, the dermatologic and venereal disease clinics, and the contagious disease wards; and lectures in tropical medicine by competent authorities are being arranged. In addition, first year students should be given an intensive course in first aid shortly after they enter medical school in order that they may do their bit intelligently, should this country be subjected to bombing raids. For the same reason, every practicing physician should complete a refresher course in first aid in order that he may be better prepared in case of enemy raids in his vicinity.

Lastly, it is obvious that organized post-graduate instruction of physicians remaining in civilian practice must of necessity be sharply curtailed for the duration of war. There are two clear-cut reasons for this: (1) the medical officers of the Army and Navy must be given first consideration in post-graduate teaching; (2) owing to the shortage of physicians left in private practice, it will be almost impossible for any individual to take time off from his duties at home to devote to post-graduate courses.

We have attempted to enumerate the major changes in medical education that have been brought about by the war. It is quite apparent that every man and woman connected in any way with medical education may expect to work harder and longer hours. Americans at Bataan, Wake, the Coral Sea, Midway, the Solomons, and Dieppe have more than done their part. It is utterly unthinkable that we of the medical profession could let them down!

W. H. B.

REVIEWS

Psychiatry in Medical Education. By FRANKLIN G. EBAUGH, M.D., and CHARLES A. RYMER, M.D. With Foreword by Dr. Adolf Meyer. 619 pages; 24 × 16 cm. The Commonwealth Fund, New York. 1942. Price, $3.50.

This book will be of chief interest to psychiatric teachers in our medical colleges, although it would well repay study by medical educators in general, by psychiatrists who are interested in the present status and in the future of their specialty, and by young physicians who are interested in taking up psychiatry as a specialty. It represents an enormous amount of work in collecting a large number of facts. Much of this information was collected directly by the senior author.

The first section of the book concerns itself with general psychiatric ideology. I can recommend the chapter on Psychobiology to anyone to whom this is a more or less meaningless word. It contains one of the most lucid explanations of what psychobiology means that has so far appeared. This is followed by a careful survey of instruction offered in psychiatry in all Class-A medical schools in the United States and Canada. It is full of detail as to variations in courses and attitudes in the various schools. It winds up with a chapter on the opportunities for psychiatric education and some detailed and helpful suggestions for setting up psychiatric services in general hospitals, pointing out the great need for such services, their feasibility and usefulness to the general hospital especially in those localities where for various reasons it is not practical to establish a separate psychiatric unit, and incidentally, points out that financially such a service not only maintains itself but should add a pleasant return to the hospital's income. The next section has to do with graduate and post-graduate training in Psychiatry and does not seem quite as adequate as the other sections, but again contains a great deal of useful information as to procedure in obtaining such training and about what the candidate may expect. The last section of the book has to do with prospects for psychiatric development in the near future and a review as to what seems a desirable psychiatric curriculum in our medical schools.

The appendices take up the methods by which the information was obtained and samples of examination and case study technic as used in some of our better psychiatric centers.

I am afraid the field of the book is extremely limited. Within this field it should fill a very definite need.

H. M. M.

Clinical Cardiology. By WILLIAM DRESSLER, M.D. 692 pages; 24 × 16.5 cm. Paul B. Hoeber, Inc., New York. 1942. Price, $7.50.

Students and practitioners will find this book a useful guide to a better understanding of cardiovascular diseases. The author's intention to stress clinical methods and bedside diagnosis is effectively accomplished. The descriptions of physical signs and methods of examination are worthwhile and the attention to diagnostic aids in obscure cases is valuable. For the most part the material is well presented and in agreement with accepted beliefs. By omitting references to the literature the author has achieved better continuity of the text, and one is able to follow the subject matter with a minimum of effort.

The outstanding weakness of the book is in the organization. One finds in Part II discussions of pulsatory phenomena which might better have been included with other physical signs in Part I. The greatest need for rearrangement is in Part III

where it would be preferable to have chapters in orderly sequence according to etiology. The subject of coronary disease would be clearer if all features were presented in one chapter, and such items as pulmonary regurgitation and arteriovenous fistula should be considered with other subjects of related etiology rather than in isolated chapters. Peripheral circulatory failure and pulmonary infarction deserve fuller discussion. Improvement in organization would make the very valuable contents of this book more accessible to the reader.

C. E. L.

BOOKS RECEIVED

Books received during September are acknowledged in the following section. As far as practicable, those of special interest will be selected for review later, but it is not possible to discuss all of them.

Advances in Pediatrics. Vol. I. Edited by ADOLPH G. DESANCTIS, M.D. 306 pages; 23.5 × 16 cm. 1942. Interscience Publishers, Inc., New York. Price, $4.50.

Formulary and Handbook. The Johns Hopkins Hospital. Edited by JOHN C. KRANTZ, JR. 253 pages; 17.5 × 11.5 cm. 1942. John D. Lucas Co., Baltimore. Price, $2.00.

Roentgen Treatment of Diseases of the Nervous System. By CORNELIUS G. DYKE, M.D., F.A.C.R., and LEO M. DAVIDOFF, M.D., F.A.C.S. 198 pages; 24 × 15.5 cm. 1942. Lea and Febiger, Philadelphia. Price, $3.25.

Medical Applications of the Short Wave Current. Second Edition. By WILLIAM BIERMAN, M.D. With a Chapter on Physical and Technical Aspects by Myron M. Schwarzschild, M.A. 344 pages; 23.5 × 16 cm. 1942. Williams and Wilkins Co., Baltimore. Price, $5.00.

Introduction to Parasitology. By A. S. PEARSE, Professor of Zoölogy, Duke University. 357 pages; 23 × 14.5 cm. 1942. Charles C. Thomas, Springfield, Illinois. Price, $3.75.

A Short History of Cardiology. By JAMES B. HERRICK, M.D. 258 pages; 23 × 15 cm. 1942. Charles C. Thomas, Springfield, Illinois. Price, $3.50.

A Curriculum for Schools of Medical Technology. Second Edition, revised. By ISRAEL DAVIDSOHN, M.D. 47 pages; 28 × 21.5 cm. 1942. Registry of Medical Technologists of the American Society of Clinical Pathologists, Ball Memorial Hospital, Muncie, Indiana. Price, $1.75.

Medical Progress Annual. Vol. III. By ROBERT N. NYE, M.D. 678 pages; 24 × 16 cm. 1942. Charles C. Thomas, Springfield, Illinois. Price, $5.00.

Human Pathology. Sixth Edition. By HOWARD T. KARSNER, M.D. 817 pages; 26 × 18.5 cm. 1942. J. B. Lippincott Co., Philadelphia. Price, $10.00.

First Aid—Surgical and Medical. By WARREN H. COLE, M.D., F.A.C.S., and CHARLES B. PUESTOW, B.S., M.S., M.D., Ph.D., F.A.C.S. 351 pages; 22 × 15 cm. 1942. D. Appleton-Century Co., Inc., New York. Price, $3.00.

COLLEGE NEWS NOTES

More A. C. P. Members Serving in the Armed Forces

Below appear the names of fifty-four additional members of the College who are serving in the armed forces of their country. Published in the July number of this journal were the names of 667, in the September number 147, and in the October number 105, making a total reported to date (October 8, 1942) of 973, more than 20 per cent of the College membership of 4,808, Masters, Fellows and Associates combined. This number on active military service from the College is especially impressive when it is remembered the average age of members of the College is definitely much higher than the average age of physicians at large. Six members of the College have already been reported "missing in action."

Ellery G. Allen
Benjamin Ashe

Frederick R. Bailey
Gerald A. Beatty
William G. Bernhard
Michael Bernreiter
James M. Bethea
Theodore L. Bliss
James M. Bowers

Richard R. Dalrymple
Perk L. Davis
Frank S. Dietrich
Mark S. Dougherty, Jr.
Thomas J. Dry

Donald R. Ferguson
Robert F. Foster
Joseph J. Furlong

Cleo R. Gatley
William H. Gordon
James R. Gudger

James A. Halsted
Joseph M. Hayman, Jr.
William R. Hewitt
W. Paul Holbrook
William L. Howell

Louis Jaffe

Newton A. Kilgore, Jr.
John T. King
Robert L. King
Phillip T. Knies

Frederick Lemere

George G. Martin
Marsh McCall
Sylvester McGinn
Richard M. McKean
Samuel Morrison

Robert Clinton Page
Robert Collier Page
*Robert T. Phillips
Harry H. Pote

John A. Reisinger
George P. Robb
Max H. Rosenblum

Edward W. Schoenheit
Howard B. Sprague
Harold Sugarman

Myer Teitelbaum
John W. Torbett, Jr.
Wilmot C. Townsend

Neville T. Ussher

Samuel A. Vogel

Robert J. Williams
Olin G. Wilson
Alfred M. Wolfe

* Missing in action.

Gifts to the College Library

We gratefully acknowledge receipt of the following gifts to the College Library of Publications by Members:

Reprints

Dr. Archie H. Beard, F.A.C.P., Minneapolis, Minn.—4 reprints;
Dr. Nathan Blumberg, F.A.C.P., Philadelphia, Pa.—1 reprint;
Dr. J. Bailey Carter, F.A.C.P., Chicago, Ill.—1 reprint;
Dr. Guy H. Faget, F.A.C.P., Carville, La.—1 reprint;
Dr. James M. Flynn, F.A.C.P., Rochester, N. Y.—1 reprint;
Dr. Hyman I. Goldstein (Associate), Camden, N. J.—1 reprint;
Dr. Wybren Hiemstra, F.A.C.P., Warren, Pa.—1 reprint;
Dr. Charles E. Lyght, F.A.C.P., Northfield, Minn.—1 reprint;
Bert E. Mulvey, F.A.C.P., Major, (MC), U. S. Army—1 reprint;
Robert Collier Page (Associate), Captain, (MC), U. S. Army—1 reprint;
Dr. Franklin B. Peck, F.A.C.P., Indianapolis, Ind.—1 reprint;
Dr. Meyer S. Rednick, F.A.C.P., Ossining, N. Y.—1 reprint;
Dr. Lea A. Riely, F.A.C.P., Oklahoma City, Okla.—1 reprint;
Dr. Leon Schiff, F.A.C.P., Cincinnati, Ohio—1 reprint;
Dr. Henry A. Tadgell (Associate), Boston, Mass.—3 reprints.

American College of Physicians to Extend Its Activities

With the development of the war situation, the Board of Regents of the College will sponsor an extension of regional scientific and social meetings, and will ask the Advisory Committee on Postgraduate Courses to formulate an extended scientific program not only for the College membership, but postgraduate seminars for Army and Navy physicians.

On October 20, 21 and 22, the College conducted a series of "Postgraduate Nights" for the naval medical personnel of the U. S. Naval Hospital, Philadelphia, extending the invitation to all medical officers of the armed forces, to physicians anticipating early entry on active duty and to members of the College. The program was prepared by Dr. Thomas M. McMillan, F.A.C.P., Philadelphia, in collaboration with Lt. Comdr. Edward L. Bortz, F.A.C.P., Chairman of the Advisory Committee on Postgraduate Courses of the College and College Governor for Eastern Pennsylvania, with Captain Richard H. Laning, Commanding Officer of the U. S. Naval Hospital, Philadelphia, and with Lt. Comdr. Gordon B. Tayloe, Chief of Medicine at the same institution; the program being as follows:

Tuesday, October 20, 1942

8:00 p.m.

GASTRO-INTESTINAL PROBLEMS

"A Classification of Chronic Diarrhea. A Discussion of the Alvine Bloody Fluxes." Henry L. Bockus, M.D., F.A.C.P., Professor of Gastro-enterology, University of Pennsylvania Graduate School of Medicine, Philadelphia.

"Recent Advances in Our Knowledge of Small Bowel Diseases." Charles L. Brown, M.D., F.A.C.P., Professor of Medicine and Head of Department of Medicine, Temple University School of Medicine, Philadelphia, Pa.

"Functional Gastro-intestinal Disturbances." Joseph C. Yaskin, M.D. (by invitation), Professor of Neurology, University of Pennsylvania Graduate School of Medicine, Philadelphia, Pa.

Wednesday, October 21, 1942

8:00 p.m.

BLOOD STUDIES

"Toxic Effects of the Sulfonamides on the Blood." Harrison F. Flippin, M.D., F.A.C.P., Associate in Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pa.

"Importance of Blood Examination in Certain Tropical Diseases." W. Harding Kneedler, M.D. (by invitation), Associate in Medicine, Jefferson Medical College, Philadelphia, Pa.

"The Management of Purpura." George E. Farrar, Jr., M.D., F.A.C.P., Assistant Professor of Medicine, Temple University School of Medicine, Philadelphia, Pa.

Thursday, October 22, 1942

8:00 p.m.

SOME OF THE MEDICAL ASPECTS OF TRAUMA

"The Crush Syndrome and Burns." Walter Estell Lee, M.D., F.A.C.S. (by invitation), Professor of Surgery, University of Pennsylvania Graduate School of Medicine, Philadelphia, Pa.

"Problems of Fluid Balance in the Traumatized Patient." Jonathan E. Rhoads, M.D. (by invitation), Associate in Surgery, University of Pennsylvania Graduate School of Medicine, Philadelphia, Pa.

"Post-Transfusion Reactions (Blood, Serum and Plasma)." Max Strumia, M.D. (by invitation), Assistant Professor of Pathology, University of Pennsylvania Graduate School of Medicine, Philadelphia, Pa.

The Fifth Annual Round-Up, or Regional Meeting, October 23, for members of the College of Eastern Pennsylvania culminated the program with a buffet luncheon at the College Headquarters in Philadelphia, followed by a scientific program at the U. S. Naval Hospital, Philadelphia, and an evening dinner-meeting addressed by Rear Admiral Ross T. McIntire, M.D., F.A.C.P., the Surgeon General of the U. S. Navy.

The afternoon program was as follows:

Auditorium, U. S. Naval Hospital

1. "35 mm. Films in the Diagnosis of Chest Conditions." Edmund C. Boots, M.D., F.A.C.P., Medical Supervisor, School Health Service, Pittsburgh Board of Public Education; Associate Staff, Columbia Hospital; Pittsburgh, Pa.
2. "Carcinoma of the Lung." Ferdinand Fetter, M.D., F.A.C.P., Lieutenant Commander, Medical Corps, U. S. Naval Reserve; Associate in Medicine, University of Pennsylvania School of Medicine.
3. "Management of Liver Disease." William S. McCann, M.D., F.A.C.P., Dewey Professor of Medicine, University of Rochester School of Medicine; Physician-in-Chief, Strong Memorial and Rochester Municipal Hospitals; Rochester, N. Y.
4. "Fever Therapy." Gordon B. Tayloe, M.D., Lieutenant Commander, Medical Corps, U. S. Navy; Chief of Medicine, U. S. Naval Hospital, Philadelphia, Pa.
5. "Peptic Ulcer in the U. S. Navy." Victor W. Logan, M.D., F.A.C.P., Lieutenant Commander, Medical Corps, U. S. Naval Reserve; Assistant Attending Physician and Gastro-enterologist in Outpatient Department, Roosevelt Hospital, New York City.

6. "Neurotic Reaction in War." F. H. Lewey, M.D., Professor of Neurophysiology, University of Pennsylvania School of Medicine; Professor of Neuropathology, University of Pennsylvania Graduate School of Medicine; Philadelphia, Pa.

At the evening meeting, among the distinguished guests were: Dr. James E. Paullin, F.A.C.P., President of the College, Atlanta, Ga.; Dr. Ernest E. Irons, F.A.C.P., President-Elect of the College, Chicago, Ill.; Dr. William B. Breed, F.A.C.P., Chairman of the Board of Governors of the College, Boston, Mass.; Capt. Robert E. Duncan, F.A.C.P., U. S. Naval Medical Center, Bethesda, Md.; Capt. A. H. Allen, Executive Officer, U. S. Naval Hospital, Philadelphia, Pa.; the College Governors for several surrounding States; Executive Officers of the Philadelphia County Medical Society and the College of Physicians of Philadelphia; the Philadelphia Director of Public Health, Dr. Hubley R. Owen, and the Coroner for Philadelphia, Dr. Herbert Goddard. Also present were the Deans of the six medical schools in Philadelphia. Members of the College were in attendance not only from the Eastern Pennsylvania district, but from Western Pennsylvania, New Jersey, New York, Delaware, Maryland, the District of Columbia and from some other more distant points.

Acting Governors of the College Appointed for Idaho and Maryland

Due to active military service by Dr. Louis Krause, College Governor for Maryland, the Executive Committee of the Board of Regents has appointed Dr. Wetherbee Fort, F.A.C.P., of Baltimore, as the Acting Governor for Maryland during Dr. Krause's absence and inability to serve.

Dr. Charles Henry Sprague, College Governor for Idaho, has accepted a full-time administrative appointment with the American College of Surgeons in Chicago, and Dr. Samuel M. Poindexter, F.A.C.P., Boise, has been appointed by the Executive Committee of the Board of Regents as Acting Governor for Idaho.

Annual Fall Meetings of the Committees and Regents of the College

The annual meetings of the several committees and of the Board of Regents of the American College of Physicians will be held at the College Headquarters in Philadelphia, December 12–13, 1942. It is at this meeting that the great bulk of College business is transacted, including the consideration of candidates for Associateship and for Fellowship. All proposals for membership must be submitted at least thirty days in advance of action. The next succeeding meeting of the Committee on Credentials will probably be held in March or April, 1943.

At a meeting of the Rock County (Wis.) Medical Society, September 22, in Beloit, Dr. M. Meredith Baumgartner (Associate), Janesville, spoke on "Hyperchromic Anemias" and Dr. Vincent W. Koch, F.A.C.P., Janesville, spoke on "Purpuras and Hemolytic Anemias."

Dr. Herbert T. Kelly, F.A.C.P., Philadelphia, Pa., presented a paper entitled "What Lies Ahead for the Physician in Nutrition" at a meeting of the Pennsylvania Nutrition Council in Harrisburg, September 18, 1942. Dr. Kelly has been elected Honorary Chairman of the Council for the year 1942–43.

Dr. Aaron E. Parsonnet, F.A.C.P., Newark, N. J., addressed the medical staff of the Station Hospital at Fort Dix, N. J., on Wednesday, September 30, on "The DaCosta Syndrome."

Dr. Richard M. Burke, F.A.C.P., Superintendent of the Western Oklahoma Tuberculosis Sanatorium, Clinton, Okla., has resigned. He plans to enter private practice in Oklahoma City.

The Third Annual Medical Meeting of the National Foundation for Infantile Paralysis will be held in New York, N. Y., December 3–4, 1942.

Dr. Henry B. Mulholland, F.A.C.P., has been appointed Assistant Dean of the University of Virginia Department of Medicine.

Dr. J. Edwin Wood, F.A.C.P., Acting College Governor for Virginia, University, addressed the James River Medical Society in Scottsville, Va., July 15. Dr. Wood discussed the use of certain drugs in heart disease.

Dr. Tom D. Spies, F.A.C.P., has accepted the invitation of the Medical Advisory Board of the Hillman Hospital, Birmingham, Ala., and the Jefferson County Commission to continue his experimental work in vitamin research at the Hillman Hospital for at least another year.

The Indiana State Medical Association held its 93rd Annual Session at French Lick, September 29–October 1. Among the guest speakers were:

Dr. Norman H. Plummer, F.A.C.P., New York, N. Y.—"Treatment of Pneumonia";

Dr. Lester R. Dragstedt, F.A.C.P., Chicago, Ill.—"Some Physiologic Principles in Surgery of the Pancreas";

Dr. Arlie R. Barnes, F.A.C.P., Rochester, Minn.—"Heart Disease."

Dr. James E. Paullin, F.A.C.P., President of the College, Atlanta, Ga., spoke on "American Medicine in the Present Emergency" at the annual banquet of the Association, September 30.

Dr. Morris Flexner, F.A.C.P., and Dr. Max L. Garon, F.A.C.P., both of Louisville, presented a paper on "Virus Pneumonia" at the recent annual meeting of the Kentucky State Medical Association.

The 75th Annual Meeting of the Michigan State Medical Society was held in Grand Rapids, September 23–25, 1942. Among the guest speakers were:

Dr. John A. Toomey, F.A.C.P., Cleveland, Ohio—"Chemotherapy in Childhood";
Dr. Harrison F. Flippin, F.A.C.P., Philadelphia, Pa.—"Sulfonamide Therapy in General Practice";
Dr. Roy W. Scott, F.A.C.P., Cleveland, Ohio—"Clinical Aspects of Arteriosclerosis";
Dr. Elmer L. Sevringhaus, F.A.C.P., Madison, Wis.—"Diagnostic and Therapeutic Problems of Obesity";
Dr. John B. Youmans, F.A.C.P., Nashville, Tenn.—"The Clinical Importance of Protein in the Diet";
Dr. James Burns Amberson, Jr., F.A.C.P., New York, N. Y.—"Clinical Interpretation of Early Tuberculosis";
Dr. Irvine H. Page (Associate), Indianapolis, Ind.—"The Nature and Experimental Treatment of Hypertension."

Dr. Charles F. McKhann, F.A.C.P., Ann Arbor, participated in a round table discussion on "Poliomyelitis."

George F. Lull, F.A.C.P., Colonel, (MC), U. S. Army, delivered the Biddle Oration of the Society, September 23. Col. Lull spoke on "Résumé of Military Medical Personnel Problems in the Army."

Dr. Harold G. Wolff, F.A.C.P., New York, N. Y., has been named neurologist in charge of a pavilion of twenty-nine beds at the New York Hospital for the study and treatment of neurologic cases.

The Medical Society of the State of Pennsylvania held its 92nd Annual Session in Pittsburgh, October 5–8. Among the guest speakers were:

George R. Callender, F.A.C.P., Colonel, (MC), U. S. Army—"Wound Ballistics";
Dr. John H. Foulger, F.A.C.P., Wilmington, Del.—"Important Factors in Industrial Preventive Medicine";
Dr. Russell L. Haden, F.A.C.P., Cleveland, Ohio—"Intravenous Therapy."
Dr. Augustus S. Kech, F.A.C.P., Altoona, was chosen President-Elect and Dr. Truman G. Schnabel, F.A.C.P., Philadelphia, was named Speaker of the House of Delegates.

James B. Collip, F.A.C.P., Montreal, Que., has been elected President of the Royal Society of Canada.

Among the speakers at the Clinical Congress conducted by the Connecticut State Medical Society in New Haven, September 29–October 1, were:

Dr. William W. Herrick, F.A.C.P., New York, N. Y.—"Medical Aspects of Obstetrics";
Dr. Lester M. Morrison, F.A.C.P., Philadelphia, Pa.—"Chemotherapy of the Gastro-intestinal Tract";
Dr. Maxwell Finland, F.A.C.P., Boston, Mass.—"Chemotherapy in the Bacteremias";
Dr. Francis G. Blake, F.A.C.P., New Haven, Conn.—"Use of Penicillin."

Dr. Harrison F. Flippin, F.A.C.P., Philadelphia, Pa., and Dr. Harry F. Dowling (Associate), Washington, D. C., participated in a panel discussion on "The Sulfonamides" at the 14th Annual Scientific Assembly of the Medical Society of the District of Columbia, September 29–October 1.

Dr. William F. Kendall, F.A.C.P., has been appointed Manager at the Veterans Administration Facility, Dwight, Ill.

Dr. Lay Martin, F.A.C.P., Baltimore, Md., is making a trip through Central and South America under an appointment by Nelson A. Rockefeller, Coördinator of Inter-American Affairs.

Dr. Henry A. Christian, F.A.C.P., Brookline, Mass., Hersey Professor of the Theory and Practice of Physic, Emeritus, has been invited to return to active duty to give clinical instruction, by the President and Fellows of Harvard University. Dr. Christian has also been appointed Visiting Physician at the Beth Israel Hospital, Boston.

Dr. Ivor E. Reed, F.A.C.P., Detroit, has been appointed a member of a technical advisory committee to the Michigan Crippled Children Commission.

Dr. Robert A. Peers, F.A.C.P., Colfax, Calif., spoke on "Control of Tuberculosis in the Individual Patient and Among His Contacts" at a meeting of the Nevada State Medical Association in Reno, September 24–26.

Dr. Charles H. Marcy, F.A.C.P., Pittsburgh, spoke on "Tuberculosis and the Home Front" and Dr. Herbert T. Kelly, F.A.C.P., Philadelphia, Pa., spoke on "Nutrition and the War Effort" at the 3rd Pennsylvania Health Institute, held in Harrisburg, September 28–30.

Dr. Herbert Z. Giffin, F.A.C.P., Rochester, spoke on "Miscellaneous Observations on the Diagnosis and Treatment of Anemia" and Dr. Walter S. Neff (Associate), Virginia, spoke on "Shock Associated with Burns" at the annual session of the Northern Minnesota Medical Association in Bemidji, August 29.

Under the Presidency of Dr. Jess V. Bell, F.A.C.P., Kansas City, Mo., the Kansas City Southwest Clinical Society held its annual fall conference, October 5–8, 1942. Among the guest speakers were:

Dr. Cornelius P. Rhoads, F.A.C.P., New York, N. Y.—"Nutrition and Cancer";
Dr. Byrl R. Kirklin, F.A.C.P., Rochester, Minn.—"Cancer of the Gastrointestinal Tract: Its Early Manifestations";

Dr. S. Marx White, F.A.C.P., Minneapolis, Minn.—"Management and Training for the Patient with Essential Hypertension";
Dr. Wesley W. Spink, F.A.C.P., Minneapolis, Minn.—"Chemotherapy of Infectious Diseases";
Dr. Richard H. Freyberg (Associate), Ann Arbor, Mich.—"General Management of the Patient with Rheumatoid Arthritis."

Dr. Chester S. Keefer, F.A.C.P., Boston, Mass., spoke on "Some of the Problems Concerned with the Interpretation of Abdominal Pain" at the annual meeting of the Vermont State Medical Society in Montpelier, October 1.

The Medical Society of Virginia held its annual meeting in Roanoke, October 5–7. The guest speakers at this meeting were Dr. James E. Paullin, F.A.C.P., Atlanta, Ga., President of the College, and Dr. Julian M. Ruffin, F.A.C.P., Durham, N. C. Among the Virginia physicians who participated were:

Dr. T. Dewey Davis, F.A.C.P., Richmond—"Acute Cirrhosis of the Liver";
Dr. Paul D. Camp, F.A.C.P., Richmond—"Rheumatic Fever and Rheumatic Heart Disease in Virginia";
Dr. David C. Wilson, F.A.C.P., Charlottesville—"Treatment of the Mental Diseases Related to the Involutional Period."

Under the Presidency of Dr. George R. Minot, F.A.C.P., Boston, Mass., the 27th Annual International Medical Assembly of the Inter-State Postgraduate Medical Association of North America was held in Chicago, Ill., October 26–30. Among the members of the College who spoke were:

Dr. Elmer L. Sevringhaus, F.A.C.P., Madison, Wis.—"The Male Climacteric";
Dr. Tom D. Spies, F.A.C.P., Cincinnati, Ohio—"The Use and Abuse of Vitamins."

Dr. Louis H. Clerf, F.A.C.P., Philadelphia, Pa., spoke on "Cricopharyngeal Spasm" and Dr. John A. Kolmer, F.A.C.P., Philadelphia, Pa., participated in a symposium on "Chemotherapy" at the 47th Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, held in Chicago, Ill., October 11–14, 1942.

The Joint Committee on Post-Graduate Education of the Long Island College of Medicine and the Medical Society of the County of Kings conducted postgraduate courses in the various branches of medicine, including arthritis, clinical cardiology, gastro-enterology, hematology, and diabetes. The courses began early in October.

Dr. Simon R. Blatteis, F.A.C.P., Brooklyn, is Chairman of the Joint Committee, and Dr. Jean A. Curran, F.A.C.P., is the representative of the Long Island College of Medicine.

1942 Supplement to the A. C. P. Directory Published

As of September 1, 1942, a Supplement to the 1941 Directory of the American College of Physicians was published and distributed to all members in good standing, with the exception of members on active military duty in the field. It was felt that this Supplement would be of no immediate value to these men on active duty, but the Executive Offices of the College will gladly mail a copy on request and without charge to any of these members.

The Supplement contains the names of Officers, Regents and Governors for the current year, committee personnel, new regulations, additions to Life Membership, records of awards of the Phillips Medal and of research fellowships, advancements to Fellowship during the past year and additions to the geographical and alphabetical rosters of members. In addition it has a record of members reported missing in military action, a list of deceased members and a list of those who have been removed from the roster by resignation or for other reasons.

Portraits of Early Medical Heroes Presented to Surgeons General of Army and Navy

Portraits of six early Surgeons General of the Army and Navy have recently been presented to the respective medical services of the Army and Navy by Ciba Pharmaceutical Products, Inc., of Summit, N. J. The paintings were by Ishmael, and the studies of these men who helped to found the medical services were reconstructed from all available early sketches and prints, in which the artist was aided by Col. Harold W. Jones, F.A.C.P., Librarian, Army Medical Library, and Capt. Louis H. Roddis, F.A.C.P., Editor of the Naval Medical Bulletin. Backgrounds of the paintings were suggestive of the exploits of the subjects, who were:

William Paul Crillon Barton, the first Chief of the Bureau of Medicine and Surgery of the Navy, 1786–1856.

Jonathan M. Foltz, Chief of the Bureau of Medicine and Surgery and Surgeon General, U. S. Navy, 1810–1877.

Elisha Kent Kane, Medical Officer of the U. S. Navy, 1820–1857.

Charles S. Tripler, Brigadier General, Army Medical Corps, 1806–1866.

Jonathan Letterman, Surgeon Major, U. S. Army, 1824–1872.

Bernard J. D. Irwin, Brigadier General, Army Medical Corps, 1830–1917.

Together, these men form a body of scientific pioneers who helped to establish the medical services of the Army and Navy and make them the great implement for the saving of life they are today. Barton standardized medical supplies, made of the crude sick bay a hospital and was responsible for placing a medical library in each unit. Foltz, known as Surgeon of the Seas because of his wide service, saw the transition of wooden ships to iron ones and forwarded in many ways the Navy in hygiene. Kane headed an expedition into the Arctic to seek a missing brother officer and during that trip added much to our knowledge of the treatment of scurvy. Tripler, of the Army, standardized the selection of recruits and wrote a manual which is still the basis of our standardization of the new Army men. Letterman established the first Ambulance Corps which was tested during the bloody Battle of Fredericksburg, and became standard practice in the Union Army. Letterman General Hospital in San Francisco is named in his honor. Irwin, during the Battle of Shiloh, organized the first tent hospital which has served as a model for field hospitals ever since. The site of his original hospital is marked with a Government tablet.

The presentation of the portraits of the Navy heroes was made to Rear Admiral Charles M. Oman, M.D., U. S. Navy, at the new National Naval Medical Center at Bethesda, Md., where the portraits will be hung in the library for present and future generations of the Navy physicians to see.

The presentation of the portraits of the Army Surgeons General was made in the stately old Army Medical Library. Colonel Harold W. Jones, F.A.C.P., Medical Corps, U. S. Army, accepted them from Mr. Brodbeck on behalf of the Surgeon General of the Army. The Army Medical Library contains many handsome portraits of past officers of the Army Medical Service and these three will be added to the collection.

Pay-Your-Doctor-Week

The Fifth Annual Pay-Your-Doctor-Week was observed November 1–7, in Los Angeles and several other cities. The plan, inaugurated in 1938 by the California Bank of Los Angeles, suggests to the public that they should not only pay their doctors, but also arrange to retire all outstanding debts and place themselves upon a cash basis, thus supporting the contention of the administration that to get out of debt is a patriotic duty—in fact, a necessity in order that the public may meet the increasing tax load and their responsibilities to buy more War Bonds.

The American Public Health Association held its 71st Annual Meeting in St. Louis, October 27–30. Some twenty other organizations in the public health field held meetings and conferences at the same time. The program was largely devoted to war time health problems—nutrition, industrial hygiene, the control of communicable diseases, maternal and child health and housing.

The U. S. Census Bureau has inaugurated a health program providing extensive medical facilities for its employes in its new building at Suitland, Md. Dr. Leon Schwartz (Associate), formerly of Philadelphia, but now in the U. S. Public Health Service, has been placed in charge. He will be assisted by a supervising nurse and a group of junior nurses. Necessary examining equipment, including mobile x-ray units, has been installed. The census unit will make preplacement physical examinations, supplementing those required for employment by the Civil Service Commission, and will treat illnesses or injuries of employes that develop during their time at work. However, actual treatment will be left to family physicians. Health records will be kept for each worker and studies will be carried on to prevent or reduce loss of time due to occupational and other diseases.

War Production Board Seeks Used Standard Typewriters from General Public

The War Production Board announces a need for 500,000 standard typewriters, made after January 1, 1935, for use in the Army, Navy and Government departments. Purchases based on trade-in allowance value as of February 1, 1941, will be made through typewriter manufacturers' representatives and approved dealers, who will condition the typewriter and ship it direct to the nearest Army or Navy supply depot. At the time the seller delivers his typewriter, the buying agent will issue a Procure-

ment Division Receipt and will place a decalcomania label on the typewriter, reading, "Property of the U. S. Government."

Manufacturers' records indicate the quantity and ownership of the majority of typewriters in civilian hands, which are suitable for military needs, and this appeal is made to present a fair opportunity to owners of such equipment to voluntarily assist in raising the required number of machines for sale to the Government. "A prompt response to this appeal will forestall and be preferable to any requisitioning of typing equipment on Government order, and your patriotic intentions will be measured by the coöperation and effort made to obtain the machines needed."

The American Gastroenterological Association on January 1, 1943, will publish the first issue of a new Journal to be called, GASTROENTEROLOGY. The new Journal will be owned by the Association, will be the official publication of the Association, and will be published by Williams and Wilkins Company. It will appear monthly, and the subscription price will be $6.00 per year.

Dr. W. C. Alvarez will be the Editor (after June, 1943) and Dr. A. C. Ivy will be the Assistant Editor. The Editorial Board will consist of Drs. A. H. Aaron (Buffalo), J. A. Bargen (Rochester), H. L. Bockus (Philadelphia), W. C. Boeck (Los Angeles), B. B. Crohn (New York), R. Elman (St. Louis), F. Hollander (New York), Sara Jordan (Boston), J. L. Kantor (New York), B. R. Kirklin (Rochester), P. Klemperer (New York), F. H. Lahey (Boston), F. C. Mann (Rochester), H. J. Moersch (Rochester), V. C. Myers (Cleveland), W. L. Palmer (Chicago), J. M. Ruffin (Durham), R. Schindler (Chicago), and D. L. Wilbur (San Francisco).

Gastroenterology invites for publication clinical and investigative contributions which are of interest to the general practitioner as well as to the specialist and which deal with the diseases of digestion and nutrition, including their physiological, biochemical, pathological, parasitological, radiological and surgical aspects.

Manuscripts should be sent to Dr. A. C. Ivy, *Gastroenterology*, 303 East Chicago Avenue, Chicago, Illinois. Letters regarding subscriptions and business matters should be addressed to Mr. R. S. Gill, Williams and Wilkins Company, Baltimore, Maryland.

OBITUARY

DR. WILLIAM DICK CUTTER

William Dick Cutter, Chicago, aged 63, Fellow of the American College of Physicians since 1929 and Secretary of the Council on Medical Education and Hospitals of the American Medical Association since 1931, died of coronary thrombosis at the home of his daughter in Johnson City, Tennessee, January 22, 1942.

Dr. Cutter was born in Brooklyn, New York, September 14, 1878. He attended Adelphi Academy of Brooklyn; received an A.B. degree in 1899 from Yale University, and an M.D. degree from Johns Hopkins in 1905. After graduation from Yale he served as Assistant in Biochemistry at Columbia University for two years during which time he also did graduate work in Berne, Switzerland. He served an internship in the French Hospital in New York City, and then spent five years on the hospital staff of the Copper Queen Consolidated Mining Company in Bisbee, Arizona. From 1911 to 1919 he was Professor of Pharmacology and Physiology at the University of Georgia Medical School. It was during this time that he became interested in medical education and improved standards of medical practice. He served as Secretary of the Board of Medical Examiners, New York State Board of Regents, from 1919 to 1923; then as Dean of the New York Postgraduate Medical School for five years, and for the next three years as Dean of the School of Medicine of the University of Southern California.

In 1931 he was appointed Secretary of the Council on Medical Education and Hospitals of the American Medical Association, a position to which he gave all his skill, energy and devotion for the remainder of his life. His careful compilation and study of the facts concerning various problems were essential before he expressed an opinion or reached a conclusion. He kept abreast of medical education and the problems peculiar to each medical school. Even when he was obliged to make an unfavorable ruling, the recipient felt that Dr. Cutter was fair and sympathetic. Dr. Cutter's forte was the preparation of the large and small details of numerous problems for later review by the Council.

He was a member of Phi Beta Kappa, Sigma Xi, the Association for the Advancement of Science, the Institute of Medicine of Chicago, the American Medical Association and the American College of Physicians. Since 1933 he served as Manager-editor of the Federation Bulletin of the State Medical Boards.

Dr. Cutter was a warrior ever ready to defend a principle; firm—a bit stubborn at times, honest, conservative, with faith in the slow evolution of the human race and the human individual, with a fine, mild character which at all times upheld the high position of the medical profession. We last saw him officially at the April round-up of our regional conference, happy, alert, witty, and in the company of medical men "whom he liked."

LeRoy H. Sloan, M.D., F.A.C.P.,
Governor for Northern Illinois.